

AUSTRALIAN VETERINARY EMERGENCY PLAN

AUSVETPLAN

Response strategy

Avian influenza

Version 5.3

AUSVETPLAN is a series of response plans that describe the proposed Australian approach to an emergency animal disease incident. The documents provide guidance based on sound analysis, linking policy, strategies, implementation, coordination and emergency management plans.

National Biosecurity Committee

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1 Introduction

1.1 This manual

1.1.1 Purpose

As part of AUSVETPLAN (the Australian Veterinary Emergency Plan), this response strategy contains the nationally agreed approach for the response to an incident – or suspected incident – of avian influenza (AI) in poultry or captive birds (which includes zoo birds) in Australia. It has been developed to guide decision making to ensure that a fast, efficient and effective response can be implemented consistently across Australia with minimal delay.

1.1.2 Scope

This response strategy covers AI caused by avian influenza virus.

This response strategy provides information about:

- the disease (Section 2)
- the implications for Australia, including potential pathways of introduction; social, environmental, human health and economic effects; and the critical factors for a response to the disease (Section 3)
- the agreed policy and guidelines for agencies and organisations involved in a response to an outbreak (Section 4)
- areas and premises classifications (Section 5)
- biosecurity controls, including quarantine and movement controls (Section 6)
- response surveillance and establishing proof of freedom (Section 7).

The key features of AI are described in the **Avian influenza fact sheet** (Appendix 1).

1.1.3 Development

The strategies in this document for the diagnosis and management of an outbreak of AI are based on risk assessment. They are informed by the recommendations in the World Organisation for Animal Health (WOAH) *Terrestrial animal health code* (Chapter 10.4) and the WOA *Manual of diagnostic tests and vaccines for terrestrial animals* (Chapter 3.3.4). The strategies and policy guidelines are for emergency situations and are not applicable to policies for imported animals or animal products.

1.2 Other documentation

This response strategy should be read and implemented in conjunction with:

- other AUSVETPLAN documents, including the operational, enterprise and management manuals; and any relevant guidance and resource documents. The complete series of manuals is available on the Animal Health Australia website¹
- relevant nationally agreed standard operating procedures (NASOPs). These procedures complement AUSVETPLAN and describe in detail specific actions undertaken during a response to an incident.

¹ <https://animalhealthaustralia.com.au/ausvetplan/>

NASOPs have been developed for use by jurisdictions during responses to emergency animal disease (EAD) incidents and emergencies

- relevant jurisdictional or industry policies, response plans, standard operating procedures and work instructions
- relevant Commonwealth and jurisdictional legislation and legal agreements (such as the Emergency Animal Disease Response Agreement – EADRA²), where applicable.

1.3 Training resources

EAD preparedness and response arrangements in Australia

The EAD Foundation online course³ provides livestock producers, veterinarians, veterinary students, paraveterinary staff, government personnel and emergency workers with foundation knowledge for further training in EAD preparedness and response in Australia.

Additional online training resources are available through Animal Health Australia.^{4,5}

² <https://animalhealthaustralia.com.au/eadra/>

³ <https://animalhealthaustralia.com.au/online-training-courses/>

⁴ <https://animalhealthaustralia.com.au/online-training-courses/>

⁵ <https://animalhealthaustralia.com.au/increasing-member-response-capability/>

2 Nature of the disease

Avian influenza (AI) is a highly contagious viral infection, primarily of avian species.

AI virus infections vary in pathogenicity. Infections are classed as being low pathogenicity AI (LPAI) or high pathogenicity AI (HPAI) (see Section 2.1). Clinical manifestations vary with the subtype and strain of virus, the species infected, and the presence of other diseases.

From 1955, the disease was known as either ‘virulent avian influenza’ or ‘fowl plague’. An international meeting on AI in 1984 recommended that the name ‘highly pathogenic avian influenza’ be used to describe the most pathogenic form of infection. As of 2020, the nomenclature endorsed by World Organisation for Animal Health (WOAH) is ‘high pathogenicity avian influenza’.

Changes in understanding of the epidemiology of AI virus infections, especially the capacity of some AI viruses to infect humans and the potential risk this poses for the emergence of human-to-human transfer, have led to a reassessment of the management of AI virus infections in birds.

This manual primarily addresses AI infection in avian species kept in captivity – poultry (commercial), poultry (non-commercial) and captive birds (see Glossary). The following species are referred to as poultry: chickens, turkeys, guineafowl, ducks, geese, quail, pigeons, pheasants, partridges, emus and ostriches.

The manual distinguishes between infection with HPAI, LPAI of the H5 and H7 subtypes (LPAI (H5/H7)), and LPAI of other subtypes (LPAI (not H5/H7)) (see Section 2.1).

Overseas, the emergence of HPAI virus H5Nx clade 2.3.4.4b (‘x’ due to the variety of neuraminidase subtypes detected, including H5N1 and H5N8) is associated with a significant increase in the intensity, frequency and geographic range of HPAI outbreaks in wild birds, poultry, and non-human mammals. Mortalities in wild birds have been observed in a wide range of species, seen as individual bird deaths and mass mortalities. Clade 2.3.4.4b has also resulted in unprecedented morbidity and mortality events in terrestrial and aquatic mammals. At the time of writing, HPAI virus H5Nx clade 2.3.4.4b lineage has not been detected in Australia.

2.1 Aetiology

2.1.1 AI virus classification

All influenza viruses are members of the family *Orthomyxoviridae*. Influenza viruses are enveloped segmented RNA viruses that are categorised into four types – A, B, C and D – based on the antigenic character of the internal nucleoprotein (NP) and matrix (M1) protein. All AI viruses belong to the influenza A type, and only influenza A viruses have been isolated from avian species; influenza viruses of the B, C and D types have never been isolated from birds. Only type A viruses have been known to cause pandemics in humans, although both A and B cause seasonal epidemics.⁶

Influenza A viruses are further divided into subtypes based on the antigenic characteristics of the haemagglutinin (HA) and neuraminidase (NA) surface glycoproteins. Each influenza virus contains one of each of these surface glycoprotein variants. A total of 18 HA subtypes (H1–H18) and 11 NA subtypes (N1–N11) have been previously described. All subtypes of influenza A viruses infect birds, except for H17-H18

⁶ [https://www.who.int/news-room/fact-sheets/detail/influenza-\(seasonal\)#:~:text=There%20are%204%20types%20of,the%20surface%20of%20the%20virus](https://www.who.int/news-room/fact-sheets/detail/influenza-(seasonal)#:~:text=There%20are%204%20types%20of,the%20surface%20of%20the%20virus)

combinations which have only been found in bats (Wu et al 2014). More recently, the H19 HA subtype has been identified (Fereidouni et al 2023, Karakus et al 2025).

Influenza B viruses and subtypes of influenza A virus are further characterised into many different clades and strains. New strains of influenza viruses appear and replace older strains through genetic changes and changes to their antigenic properties. New strains are named as per the World Health Organisation (WHO) convention on nomenclature for influenza viruses.⁷ Influenza A viruses have a particular tendency towards reassortment of genetic material, which can occur when two influenza viruses infect the same host cell simultaneously, resulting in a rapid shift.

2.1.2 Pathotypes

The pathogenicity of AI virus infection in one species may not necessarily reflect pathogenicity in other species. AI viruses are classified⁸ into two pathotypes – HPAI and LPAI viruses – based on either molecular characteristics or the pathogenicity of the virus in experimentally inoculated chickens.

2.1.2.1 HPAI virus

Criteria adopted by the WOAHP for determination of the pathogenicity of an influenza A virus:

- *In vivo*: One of two methods to determine pathogenicity in chickens is used. HPAI viruses either have a mean intravenous pathogenicity index (IVPI) greater than 1.2 in ten 4–8-week-old specific pathogen free (SPF) chickens, or alternately cause at least 75% mortality in eight 4–8-week-old chickens following intravenous inoculation of infective allantoic fluid.
- *In vitro*: the AI virus can be sequenced to determine the sequence of amino acids present at the cleavage site (HA0) of the HA molecule. The sequence is compared with those of other HPAI isolates. If the amino acid motif is identical to that of previous HPAI virus isolates, the isolate being tested should be considered to be HPAI virus. Any isolate with a new motif must be tested *in vivo* by IVPI.

2.1.2.2 LPAI virus

LPAI viruses are all influenza A virus subtypes that are not HPAI viruses (as defined above). The cleavage site of all H5 and H7 viruses with low pathogenicity in chickens should be determined.

2.1.3 Pathogenicity

Pathogenicity depends on both the genetic properties of the virus and the host species. In avian species (poultry and wild birds), HPAI viruses can cause severe clinical disease. Even LPAI virus subtypes can be associated with severe clinical disease in the presence of other infectious agents (e.g. infectious bronchitis virus, infectious laryngotracheitis virus). Some H5 and H7 LPAI viruses have the capability to mutate into HPAI virus.

The HA gene, specifically the amino acid sequence at the cleavage site (HA0), is the primary determinant of pathogenicity of AI viruses in chickens. The sequence of amino acids at the HA cleavage site determines the HA protein structure and the ability of protease enzymes to cleave the HA protein into two proteins (HA1 and HA2); this cleavage is essential for the virus to replicate and attain high infectivity.

⁷ www.ncbi.nlm.nih.gov/pmc/articles/PMC2395936/pdf/bullwho00427-0070.pdf

⁸ www.woah.org/fileadmin/Home/eng/Health_standards/tahm/3.03.04_AI.pdf

The HA cleavage site in LPAI viruses can only be cleaved by trypsin-like proteases produced by the epithelial cells of the respiratory and intestinal tracts. Infection with, and replication of, LPAI viruses are limited to these mucosal regions.

H5PAI viruses have an HA cleavage site that is recognised by both trypsin-like and ubiquitous (e.g. furin) proteases. This enables viral replication within many tissue and cell types.

Other factors such as the binding between the HA and the host cell receptor are important for host specificity, and cell or tissue tropism.

2.1.4 Antigenic change

Influenza A viruses have the capacity for rapid antigenic change through mutation and reassortment.

Antigenic drift is the fixation, by natural selection, of mutations in the HA and NA proteins that enable the virus to evade the host immune response, particularly in humans but also in poultry. Hence, antigenic drift enables escape from antibody-mediated neutralisation acquired following prior infection or vaccination. The underlying mechanism is the high mutation rate of influenza A viruses because of their error-prone replication. Most mutations do not confer any antigenic changes; mutations in the HA1 region of the HA protein, particularly in or around the receptor-binding site, are thought to drive antigenic evolution. The high mutation rates measured in influenza A viruses are evidenced by ladder-shaped phylogenies. Overall, antigenic drift appears to follow a 'punctuated' pattern, with major jumps occurring every 1–5 years, correspondent with poor vaccine efficacy in humans (Wille & Holmes 2020).

Antigenic shift is the rapid phenotypic change following reassortment (a special case of recombination) of intact gene segments between viruses coinfecting the same host cell. The consequence of reassortment is that coinfection allows viral progeny to contain various gene segment combinations from the different parental viruses. Reassortment is of evolutionary importance because it creates new genomic constellations. Although few of these will benefit the virus, some may facilitate adaptation to new hosts, help evade host immune responses, and assist in the generation of antiviral resistance (Wille & Holmes 2020). Because of reassortment, many different genome constellations may be detected despite an identical HA sequence; however, these different constellations may not confer antigenic change.

2.2 Susceptible species

2.2.1 Birds

Influenza A viruses have been isolated from most major bird orders. To date, over 500 bird species, of which more than half are newly affected since 2021, have been reported to be affected by HPAI.^{9, 10} LPAI viruses occur naturally in wild birds (particularly waterbirds and shorebirds) including in Australia.

When chickens and turkeys are infected with viruses with HA and NA combinations that have not occurred previously in these populations, a proportion of the viruses may mutate and produce strains that cause severe disease. Epidemics largely occur when a H5 or H7 LPAI virus has been introduced to a naive poultry population and then mutates to HPAI virus.

Globally, nearly all the possible HA and NA subtype combinations have been detected in wild birds. Species in the orders Anseriformes (ducks, geese, swans) and Charadriiformes (shorebirds, waders, gulls) are regarded as important reservoir hosts and disseminators of LPAI viruses. Avian species other than those identified as reservoirs of LPAI virus may have a higher clinical susceptibility to AI virus. For example, black swans have numerous unique genetic characteristics that are likely to result in this species being highly susceptible to viral infections such as HPAI¹¹ (Karawita et al 2023).

Since its emergence in 2020, H5Nx clade 2.3.4.4b has infected and caused disease in a wide range of avian species including poultry and wild birds overseas. These strains significantly differ from previous HPAI H5 viruses in their increased outbreak frequency, geographic range and ability to spread via a wide range of avian species including both poultry and wild birds.

AI viruses are being detected in a growing number of bird orders, including those listed in Table 2.1.¹²

⁹ www.fao.org/animal-health/situation-updates/global-aiv-with-zoonotic-potential/bird-species-affected-by-h5nx-hpai/en

¹⁰ A working list of Australian birds is available at <https://birddata.birdlife.org.au/whats-in-a-name>

¹¹ <https://wahis.woah.org/#/in-review/3332?reportId=18800&fromPage=event-dashboard-url>

¹² Sources include: <https://datazone.birdlife.org/species/taxonomy>,

https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1128143/hpai-europe-number38-230104.pdf;

www.ecdc.europa.eu/sites/default/files/documents/ON_5141v3.pdf; www.fao.org/animal-health/situation-updates/global-aiv-with-zoonotic-potential/bird-species-affected-by-h5nx-hpai/en

Table 2.1 AI virus detections in birds

Bird order	Common name¹³
Accipitriformes	Osprey, hawks and eagles
Anseriformes	Magpie Goose, ducks, geese and swans
Caprimulgiformes	Frogmouth, nightjars, owlet-nightjars, swifts
Cathartiformes	New World vultures (e.g. condors, turkey vulture)
Charadriiformes	Shorebirds
Ciconiiformes	Storks
Columbiformes	Doves and pigeons
Coraciiformes ¹⁴	Bee-eaters and kingfishers
Cuculiformes ¹⁵	Cuckoos and koels
Falconiformes	Falcons
Galliformes	Chickens, turkeys, pheasants, brush turkeys, scrubfowl and quail
Gaviiformes	Loons/divers
Gruiformes	Rails, gallinules, coots and cranes
Otidiformes ¹⁶	Bustards
Passeriformes	Passerines (perching birds)
Pelecaniformes	Ibis, herons and pelicans
Phoenicopteriformes	Flamingos
Piciformes	Jacamars, puffbirds, toucan, woodpeckers
Podicipediformes	Grebes
Procellariiformes	Fulmars, petrels, prions and shearwaters
Psittaciformes	Parrots and cockatoos
Pterocliiformes ¹⁷	Sand grouse
Sphenisciformes	Penguins
Strigiformes	Typical owl and barn owls
Struthioniformes	Ostrich, emus and cassowary
Suliformes	Gannets, boobies and cormorants
Trogoniformes	Trogons

¹³ Common names adapted from: del Hoyo and Collar (2014) HBW and BirdLife International Illustrated Checklist of the Birds of the World. Volume 1 – Non-passerines. Lynx Editions, Barcelona. (Courtesy of the Australian Department of Environment).

¹⁴ Desvaux et al 2009

¹⁵ Caron et al 2016

¹⁶ Khan et al 2009

¹⁷ Caron et al 2016

2.2.2 Non-human mammals

The ecology of AI viruses is complex. Wild birds are the main natural reservoir hosts from which AI viruses can emerge to infect non-human mammals. These spillover events of avian-origin viruses can give rise to host-adapted viruses (De Marco et al 2023).

Host-adapted viruses can be sporadic or become endemic. The avian-derived lineage of canine influenza has now circulated for 15 years (Chen et al 2023). Pigs are a reservoir for mammalian influenza viruses and have been implicated in the generation of human pandemic viruses (Smith et al 2009). This is driven by the presence of both avian and mammalian-type sialic acid receptors (Nelli et al 2010). Cases of inter-species transmission identified between birds and swine included strains of H5N1, H9N2, H4N1, H4N6, H5N3, H10N5, H4N8, H6N6, H7N9 in China; H4N6, H6N2, H7N2 in the United States of America (USA); H4N6, H3N3 in Canada; H7N2, H5N2 in South Korea; H5N1 in Nigeria and H5N1, H5N2, H9N2 in Egypt (Chauhan & Gordon, 2020). One of the established swine influenza lineages (Eurasian avian-like swine H1N1) is of avian origin (Pensaert et al 1981), demonstrating the capacity for AI to adapt and establish in swine.

AI viruses are being detected in a growing number of mammalian families, including those listed in Table 2.2. Since the emergence of the HPAI virus H5Nx clade 2.3.4.4b in 2020, unprecedented morbidity and mortality events in terrestrial and aquatic mammals have occurred, with more than 100 species known to have been affected by HPAI virus H5Nx. Affected species are mostly predatory or scavenging species with exposure likely to have occurred through close contact, contaminated environments or consumption of infected wild birds. Species include wild terrestrial and aquatic mammals, such as foxes, seals and dolphins, and domesticated mammals such as farmed American mink.¹⁸

The range of non-human mammals susceptible to infection with avian influenza H5Nx clade 2.3.4.4b continues to expand, with new reports emerging regularly. It is important not to assume that the current list of affected species in this manual is exhaustive. Further consideration should be given during an outbreak to the potential for infection in other non-mammalian species.

¹⁸ www.eurosurveillance.org/content/10.2807/1560-7917.ES.2023.28.3.2300001

Table 2.2 AI virus (natural infection) detections in non-human mammal species

Mammalian Family	Common name(s)
Balaenopteridae ^a	Rorquals
Bovidae	Cattle, goats etc
Camelidae	Camelids
Canidae	Canines (e.g. dog, fox, coyote)
Cervidae	Deer
Cricetidae	Musrat, vole, hamster, etc.
Delphinidae	Dolphins
Didelphidae	Opossums
Equidae	Horses, donkey, zebra, etc.
Felidae	Felines (e.g. cat, lynx, tiger)
Hyaenidae	Hyaena
Leporidae	Rabbits and hares
Mephitidae	Skunks
Muridae	Mice, rats, gerbils, etc.
Mustelidae	Mustelids
Ochotonidae	Pika
Odobenidae	Walrus
Otariidae	Fur seals and sea lions
Phocidae	True seals
Phocoenidae	Porpoises
Procyonidae	Raccoons
Sciuridae	Squirrels
Suidae	Pigs
Ursidae	Bears
Viverridae	Civet, genet

^a One report from Lvov et al 1978

Influenza A (IA) viruses have been recently detected in Central and South American bats (H17N10 and H18N11 subtypes) and Egyptian fruit bats (a novel H9N2-like virus). It is unknown whether these viruses originated from a bat or bird ancestor and while they are all distinct from conventional IA viruses, the novel H9N2-like virus shares several avian IA viruses characteristics. To date all three have been detected only in bats, suggesting evolution in bats for a substantial amount of time and the possibility of other unknown circulating IA viruses in bat populations (Tong et al 2012, Tong et al 2013, Kandeil et al 2019, Yang et al 2021).

AI viruses can experimentally infect a range of species, including dogs, foxes, cattle, pigs, ferrets, rats, rabbits, guinea pigs, mice, cats, mink, non-human primates and humans (Swayne & Halvorson 2003, Rimmelzwaan et al 2006, Kalthoff et al 2008).

2.2.2.1 Farm animals

In March 2024, the USA reported post mortem isolation of H5N1 clade 2.3.4.4b virus from the brain and tissues of young goats. The goats shared their living area, including feeding and drinking, with domestic poultry that had been recently depopulated due to an outbreak of HPAI.

Subsequently, multiple herds of dairy cattle in a number of states of the USA were confirmed to be infected with H5N1 clade 2.3.4.4b. The cattle infected with influenza displayed a range of non-specific clinical signs such as fever, lethargy, reduced food intake, decreased production and thick, discoloured milk.¹⁹

In May 2024, concurrent to the detections of HPAI in dairy cattle, HPAI H5N1 was detected in alpacas from a premises where HPAI-affected poultry were depopulated. The viral genome sequence was determined to be the same as that circulating in dairy cattle at the time (B3.13) and was consistent with sequences from the depopulated poultry on the premises.²⁰

Additional incidents include neurological signs and death of cats that have ingested raw colostrum and/or milk from H5N1 infected cattle and sporadic infections in dairy workers (see Section 2.2.3).

In October 2024, the United States Department of Agriculture (USDA) first reported a H5N1 detection in two non-clinical pigs on a small farm of 5 pigs housed in close proximity to AI infected domestic poultry. The virus represented a new genotype, D1.2²¹, and was distinct from the D1.1 genotype reported in poultry elsewhere in the US, and the B3.13 detected in US dairy cattle. In March 2025, a D1.1 genotype was detected in dairy cattle in the USA.

Pigs have receptors for both avian and mammalian influenza strains and are therefore a potential source of viral reassortment. In 2004, China's Harbin Veterinary Research Institute identified infection of pigs on farms with H5N1 strains of avian influenza.²²

¹⁹ www.aphis.usda.gov/sites/default/files/hpai-dairy-faqs.pdf

²⁰ www.aphis.usda.gov/livestock-poultry-disease/avian/avian-influenza/hpai-detections/mammals/highly-pathogenic-avian

²¹ www.aphis.usda.gov/news/agency-announcements/usda-animal-plant-health-inspection-service-shares-update-h5n1-detection

²² www.who.int/emergencies/disease-outbreak-news/item/2004_08_25-en

2.3 World distribution

For the latest information on the distribution of internationally notifiable AI, refer to the WOAHA World Animal Health Information System.²³

2.3.1 Zoonotic potential

Humans are susceptible to infection with AI viruses. Natural exposure to H5, H6, H7, H9 and H10 subtypes has caused zoonotic disease in various forms in humans, ranging from mild or inapparent infection to death (Mostafa et al 2018).

Human infection with H5 and H7 AI viruses has led the WHO to consider whether a new pandemic human influenza virus could be derived directly from birds. There have been no cases of sustained human-to-human transmission of AI viruses to date. Further information on human infection with AI viruses can be found on the WHO website.²⁴

Circulating antibodies to H4, H5, H6, H7, H9 and H11 antigens have been detected in people in southern China in the late 1970s and 1980s, as well as more recently, indicating probable exposure to LPAI viruses (Zhou et al 1996, Chen et al 2008, Wang et al 2009).

Since March 2013, when H7N9 virus infection was first detected in humans, a total of 1,567 laboratory-confirmed human cases, including at least 615 deaths, have been reported to the WHO.²⁵ The fatality rate for H7N9 infections is approximately 40% (Zhang et al 2020).

In Australia, poultry workers were infected with LPAI H10N7 virus when they were exposed to clinically healthy birds from an infected flock during processing (Arzey et al 2012). Experimental exposure of people to H3N8, H3N2, H5N2, H6N1, H9N2, H4N8 and H10N7 isolates indicated that these isolates could infect humans, although they were generally ineffective in virus transmission (Peiris 2009).

Overseas, human infections with clade 2.3.4.4b have been rare to date and have typically only occurred in people who have had close contact with infected poultry, wild birds or dairy cattle. Clinical severity of human cases has varied widely, which is consistent with other subtypes of AI virus. Sporadic infections have occurred in dairy workers exposed to contaminated raw cows' milk or milk products who have developed mild clinical signs including conjunctivitis. Some H5N1 clade 2.3.4.4b viruses detected in mammals have carried the PB2 E627K mutation, which is associated with increased replication and adaptation in mammalian hosts. Importantly, these viruses have not acquired changes that enable efficient human-to-human transmission.²⁶

Asian-origin H9 avian influenza viruses are increasingly recognised as a zoonotic disease threat and are also associated with disease in poultry (Carnaccini & Perez 2020). Human to human transmission has not yet been documented but there are concerns about the pandemic potential of these viruses.²⁷

²³ wahis.woah.org/#/home

²⁴ www.who.int/

²⁵ www.who.int/emergencies/disease-outbreak-news/item/05-september-2018-ah7n9-china-en

²⁶ www.fao.org/animal-health/news-events/news/detail/ongoing-avian-influenza-outbreaks-in-animals-pose-risk-to-humans/

²⁷ [www.who.int/publications/m/item/updated-joint-fao-who-woah-public-health-assessment-of-recent-influenza-a\(h5\)-virus-events-in-animals-and-people_apr2025](http://www.who.int/publications/m/item/updated-joint-fao-who-woah-public-health-assessment-of-recent-influenza-a(h5)-virus-events-in-animals-and-people_apr2025)

2.3.2 Distribution outside Australia

Subtypes of AI virus occur on all continents where research has been carried out.

Outbreaks of HPAI in North America (H5N2 in the USA in 1983 and Mexico in 1994, and H7N3 in Canada in 2004 and 2007), Italy (H7N1 in 1999), the Netherlands (H7N7 in 2003) and Chile (H7N3 in 2002) all initially involved mutation of LPAI viruses, and AI virus infection became widespread. In 1997, a novel H5N1 virus caused fatal disease in poultry and humans in Hong Kong; disease subsequently appeared in other birds and poultry throughout Asia and spread across Eurasia and into Africa. Before the emergence of this strain, AI was considered a sporadic high-mortality poultry disease.

In December 2014 and January 2015, the Animal and Plant Health Inspection Service of the USDA reported the presence of HPAI (H5N2) and HPAI (H5N8) viruses in wild birds in a few states. In January 2015, an HPAI (H5N1) virus was detected in a wild duck in the USA. The H5N1 virus isolated from the wild duck was a new mixed virus (a 'reassortant') that is a different clade from the Asian avian H5N1 viruses that have caused human infections with high mortality in several other countries (notably in Asia and Africa). No human infections with this new reassortant H5N1 virus have been reported.

In 2021-22, the European Food Safety Authority (EFSA) recorded its (then) highest incidence of HPAI infection in poultry, captive and wild birds (~5,300 detections). The persistence of the virus in wild birds indicated that the disease may have become more endemic in those species. By December 2022, HPAI cases had continued to rise among wild bird populations but had gradually declined in poultry. In March 2024, HPAI cases were still widespread, but declining across all bird types (wild, captive and domestic).²⁸ However, H5N1 HPAI infection is now considered to be endemic in poultry in some countries including Bangladesh, China, Egypt, India, Indonesia and Vietnam.

Since 2020, there has been an increase in frequency and geographic spread of HPAI outbreaks – with unprecedented impacts on poultry, wild birds and mammals – associated with HPAI H5Nx 2.3.4.4b.²⁹ HPAI H5Nx 2.3.4.4b emerged in 2020 and rapidly spread across many parts of Africa, Asia and Europe. In late 2021, HPAI H5Nx 2.3.4.4b viruses were detected in North America and subsequently spread throughout the Americas. By the end of 2022, 67 countries in five continents reported HPAI H5N1 outbreaks in poultry and wild birds to WOA, with more than 131 million domestic poultry lost due to death or culling (WOAH 2023). In late 2023 and early 2024 respectively, wildlife on Antarctic islands and mainland Antarctica were also affected by HPAI H5Nx 2.3.4.4b³⁰ in what remains an ongoing dynamic situation with expected further spread. Reassortment of genetic material with local LPAI viruses has occurred and is likely to continue.³¹

For further information on reported AI outbreaks, refer to the WOA and the Food and Agriculture Organization of the United Nations³² websites.

²⁸ www.efsa.europa.eu/en/topics/topic/avian-influenza

²⁹ www.cms.int/en/publication/h5n1-high-pathogenicity-avian-influenza-wild-birds-unprecedented-conservation-impacts; www.fao.org/animal-health/situation-updates/global-aiv-with-zoonotic-potential/en

³⁰ www.bas.ac.uk/media-post/first-confirmed-cases-of-avian-influenza-in-the-antarctic-region/

³¹ www.cms.int/sites/default/files/publication/avian_influenza_2023_aug.pdf

³² www.woah.org/en/disease/avian-influenza/; www.fao.org/animal-health/animal-diseases/highly-pathogenic-avian-influenza/en

2.3.3 Occurrence in Australia

2.3.3.1 Commercial poultry

2.3.3.1.1 HPAI

Eight outbreaks of HPAI occurred in Australia between 1976 and 2020,³³ most likely all due to LPAI viruses of Australian lineage being passed from wild waterfowl (likely ducks) to commercial poultry, followed by mutation to HPAI viruses. HPAI viruses caused clinical disease in commercial poultry in Victoria in 1976 (H7N7), 1985 (H7N7), 1992 (H7N3) and 2020 (H7N7); in Queensland in 1994 (H7N3); and in New South Wales in 1997 (H7N4), 2012 (H7N7) and 2013 (H7N2)³³ (Swayne & Suarez 2000, Scott et al 2020). Each time, severe disease occurred in affected chicken flocks. All instances had some evidence of contact with wild waterfowl or surface water contaminated by wild waterfowl, or an association with free-range farmed ducks (Westbury 1998, Scott et al 2020). There is some evidence that, initially, LPAI may have been involved in the outbreaks in 1976 (Westbury 1998), 1992 (Victorian Department of Agriculture, pers comm, 2022) and 1997 (Selleck et al 2003).

Full genome sequencing of 11 Australian H7 isolates suggested that Australian H7 isolates form a monophyletic clade (i.e. descended from a single common ancestor) when compared with H7 subtypes worldwide (Bulach et al 2010, Wille et al 2022).

In August 2020, 3 different strains of AI virus were identified across 7 infected properties in Victoria. One strain was HPAI, and 2 strains were LPAI. The HPAI infected properties were 3 free-range and cage egg farms and one composting property within the same shire (HPAI (H7N7)). Genomic sequence analysis showed that the virus was most closely linked to Australian lineage H7 LPAI viruses previously detected in wild birds and that samples collected on all three affected egg farms were genetically similar and had identical H7 sequences and the same high pathogenicity haemagglutinin (HA) cleavage site. Extensive tracing did not identify links between the first affected egg farm and the other 2 egg farms, the latter being linked by shared equipment and personnel

Until 2024, this was the largest recorded outbreak response to AI in Australia. Control measures included depopulation, decontamination and surveillance of the infected poultry farms, implementation of restricted and control areas, enhanced passive surveillance of the outside area and, for the first time in Australia, the implementation of a housing order for all poultry in the restricted and control areas in the shire affected with H7N7.

In May 2024, HPAI H7N3 was detected in a mixed caged/barn and free-range commercial layer poultry farm near Meredith, Victoria. Subsequent operational surveillance activities resulted in the detection of another six commercial poultry flocks within the Meredith area, with the last detection occurring in late June 2024. One infected premises was a pullet-rearing facility, and another was a duck farm producing eggs and meat birds. The majority of the remaining affected flocks were laying chickens.

Concurrently, a different strain, HPAI H7N9, was detected in a mixed caged and free-range commercial layer poultry farm near Terang, Victoria.

In June 2024, H7N8 was detected in a large free-range and barn commercial layer flock in the Hawkesbury Valley, New South Wales. The same virus was subsequently detected in a commercial layer flock in the Australian Capital Territory where spread was attributed to the movement of eggs and/or egg handling equipment off the index case. In addition, a commercial broiler flock, 3 backyard poultry flocks and 1 aviary bird flock were confirmed as infected with the same virus, all within 2.4 km of the index case. Genomic

³³ <https://agriculture.vic.gov.au/biosecurity/animal-diseases/poultry-diseases/avian-influenza-bird-flu>

sequence analysis of the virus showed that it was similar to Australian lineage H7 LPAI viruses detected in wild bird populations in Australia.

In February 2025, H7N8 was detected on 4 commercial free-range egg layer farms near Euroa in northern Victoria. This strain was different to that encountered in the 2024 detections in New South Wales and the Australian Capital Territory, and all properties were epidemiologically linked under the same ownership, within the same locality.

2.3.3.1.2 LPAI (H5 or H7)

Seven LPAI (H5 or H7) virus strains have been detected in Australian domestic poultry³⁴ (QDAF 2020, Scott et al 2020):

- An LPAI (H7N7) virus was isolated on a duck farm during investigation of an HPAI (H7N7) outbreak in chickens in Victoria in 1976. The ducks showed no signs of clinical disease.
- Antibodies to H5, H7 and other subtypes of AI viruses were detected in commercial domestic ducks during investigation of an HPAI (H7N3) outbreak in chickens in Victoria in 1992.
- LPAI (H5) antibodies were detected on a Tasmanian non-commercial, multispecies smallholding in 2006.
- An LPAI (H5N3) virus was detected in a free-range duck flock in Victoria during routine surveillance in 2012. The source of the virus could not be determined, but it is speculated that the primary source may have been wild birds, which had free access to the range area.
- An LPAI (H5N3) virus was isolated from 1 duck on a non-commercial holding in Western Australia in 2013. The positive test was an incidental finding during routine surveillance. There was no evidence of infection of in-contact birds or birds in the surrounding area.
- An LPAI (H5N2) virus was detected on 2 linked turkey farms in Victoria, during surveillance for a concurrent HPAI outbreak in 2020. One property was in the same shire as the HPAI outbreak.
- An LPAI (H7N6) virus was detected on an emu farm in Victoria following the report of an increase in morbidity and mortality in chicks. This was concurrent with the HPAI outbreak in Victoria, 2020.

2.3.3.1.3 LPAI (not H5 or H7)

The following LPAI (not H5 or H7) detections have occurred (QDAF 2020):

- Antibodies to LPAI H1, H4 and H9 (in addition to H5 and H7) subtypes were detected in ducks on a farm in Victoria in 1992.
- LPAI (H3N8) virus was detected on a multi-age commercial duck farm in Victoria in 1992.
- An LPAI (H6N4) virus was isolated from a single duck on a commercial chicken and duck property in Queensland in 2006.
- Chickens in several sheds from a property in New South Wales tested seropositive to LPAI (H6N4) in 2006.
- LPAI (H10N7) virus was detected in 2010 on a chicken farm in New South Wales, where transmission to abattoir workers during processing of the poultry was documented. Phylogenetic analysis of the full HA sequence of the virus involved in this event showed a higher degree of homology with North American H10 viruses (Arzey et al 2012) than with Australian H5, H7 and H9 isolates (Bulach et al 2010, Hansbro et al 2010).

³⁴ <https://agriculture.vic.gov.au/biosecurity/animal-diseases/poultry-diseases/avian-influenza-bird-flu>

- In April 2012, LPAI (H9N2) virus was confirmed on 2 turkey farms near the Hunter Valley in New South Wales; the source of the infection is unknown.
- In 2012, an LPAI (H4N6) virus was found in ducks of several age groups on a multi-age farm of 2400 ducks on the north coast of New South Wales.
- In 2012, an LPAI (H10N7) virus was detected in a Queensland poultry flock; the source of the infection is unknown, but it is postulated that the primary source may have been wild birds.
- In 2018, an LPAI (H1N2) virus was detected in Queensland in free-range chickens, ducks and guinea fowl (Scott et al 2020).
- In 2018, a LPAI (H4N6) virus was detected in a single chicken in a backyard flock in Pomborneit (south-west Victoria). In 2021, a LPAI (H10N3) virus was detected on an emu farm in Victoria following a report of increased morbidity and mortality in chicks. In 2023, a LPAI (H10N7) virus was detected on an emu farm in Victoria following a report of increased morbidity and mortality in chicks.
- In 2024, a LPAI (H9N2) virus was detected in a free-range broiler property in Western Australia following a report of increased morbidity and mortality in broilers. It is postulated that a particularly low rainfall period may have resulted in increased contact between wild birds and poultry.

2.3.3.2 Wild bird surveillance

LPAI viruses of many subtypes — including H1–H13, H15 and H16 — have been isolated from, or demonstrated in, a wide range of wild aquatic birds, including resident and migratory species, in well-separated locations in Australia (Arzey 2004, Haynes et al 2009, Hansbro et al 2010, Herfst et al 2012, Grillo et al 2015, Wille et al 2022, Wildlife Health Australia³⁵). The subtypes detected in wild birds vary between species, locations and years. HPAI viruses have never been detected in wild birds sampled in Australia as part of the National Avian Influenza Wild Bird Surveillance Program³⁶, coordinated by Wildlife Health Australia. HPAI virus H5Nx clade 2.3.4.4b lineage has not been detected in Australia.

In general, the frequency of AI virus detection^{37,38} via polymerase chain reaction (PCR) and virus isolation from surveillance of healthy Australian wild birds is low (up to 5.4% (East et al 2008, Haynes et al 2009, Grillo et al 2015, Wille et al 2023, Xie et al 2023)) compared with other global surveillance studies of wild birds (Hansbro et al 2010, Tracey 2010, Cerda-Armijo et al 2020). Detection rates can vary greatly between sampling periods and locations (Garamszegi & Møller 2007, Grillo et al 2015,), and localised sporadic increases in the frequency of AI virus detection have been recorded in Australia (Garamszegi & Møller 2007, Grillo et al 2015, Hoyer et al 2021). Studies also demonstrate higher rates of detection in some species (e.g. 0.6% in shorebirds versus 2.5% in waterfowl and significantly higher rates in dabbling ducks than in non-waterfowl species) which is consistent with findings in other geographic regions (Hansbro et al 2010, Tracey 2010, Grillo et al 2015).

Since 2005, the National Avian Influenza Wild Bird Surveillance Program has detected LPAI H5 viruses in New South Wales, the Northern Territory, Queensland, South Australia, Tasmania, Victoria, and Western Australia. The majority of these detections were in Anseriformes species, with detections also in Charadriiformes and mixed bird species samples. LPAI H7 viruses have been detected in New South Wales, Queensland, South Australia, Tasmania, Victoria, Western Australia, and the Australian Capital Territory. The majority of these detections were in Anseriformes species, with detections also in mixed bird species samples.

³⁵ <https://wildlifehealthaustralia.com.au/Resource-Centre/Surveillance-Reports?t=2>

³⁶ <https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/Wild-Bird-Surveillance>

³⁷ Comparing rates of detection is difficult because surveillance methodologies and strategies differ between studies. It is also worth noting that frequency of detection is limited to the particular populations studied and is unlikely to represent all wild bird populations.

³⁸ <https://wildlifehealthaustralia.com.au/Resource-Centre/Surveillance-Reports?t=2>

Serological surveillance in studies of healthy wild birds in Australia has detected antibodies to AI virus in a variety of bird orders, primarily in Charadriiformes and Anseriformes species but low levels of seropositivity has also been recorded in other bird orders including Passeriformes, Procellariiformes, Gruiformes and Columbiformes (Curran 2012, Wille et al 2019, Wille et al 2023).

2.4 Epidemiology

2.4.1 Incubation period

The incubation period and infectious period depend on the virus strain, the dose and route of exposure, the species exposed, the age of individuals, and the environment.

Incubation periods are extremely variable, from a few hours in intravenously inoculated birds, to 3 days in naturally infected individual birds, and up to 14 days in a flock (Swayne et al 2020).

2.4.1.1 WOAH incubation period

For the purposes of the WOAH *Terrestrial animal health code*, the incubation period³⁹ for AI at the flock level is 14 days.

2.4.2 Persistence of agent and modes of transmission

Appendix 2 provides information on infective dose and virus shedding, and Appendix 3 information on virus viability.

AI viruses are shed in the faeces and respiratory secretion of birds. Outbreaks in domesticated birds are initiated by direct and indirect contact with infected birds, including contamination of food, water, litter material and free-range pasture. The infection is then spread through the movements of infected live birds or contaminated eggs/egg products, feed, equipment, vehicles, litter/waste materials, clothing and footwear. Wild birds, infected backyard poultry and live bird markets can be sources of AI virus for commercial poultry.

In past outbreaks worldwide, dissemination of AI virus between flocks has been primarily attributed to poor biosecurity, involving:

- movement of infected birds (including vaccinated birds)
- viral contamination of water
- live bird markets (movement of birds, unsold birds returning to farm, contaminated crates and vehicles)
- human-associated movements, such as transporting food, personnel, equipment, vehicles and waste out of premises that are contaminated with infected faeces or respiratory secretions (fomites)
- centralised egg handling facilities and equipment, particularly shared use of egg trays and fillers.

Recent HPAI outbreaks in poultry (outside Australia) due to H5Nx clade 2.3.4.4b have involved direct and repeated spread from wild birds, driving both episodic resurgence and multinational transmission (Bellini et al 2022, Nagy et al 2023, Xie et al 2023). The situation is distinct from previous instances, where mortality due to HPAI virus was largely limited to poultry and spread between properties was largely facilitated by such biosecurity deficits as outlined above. In addition, the northern hemisphere influenza seasons 2020/21 and 2021/22 overlapped, due to infection levels in wild birds persisting throughout the summer of 2021 with associated mortality (Pohlmann et al 2022, Ramey et al 2022). In Europe, the maintenance of HPAI virus in wild bird populations has resulted in multiple transmissions to poultry and poultry outbreaks with parallel sources of infection (Nagy et al 2023).

³⁹ In the WOAH *Terrestrial animal health code*, 'incubation period' means the longest period that elapses between the introduction of the pathogenic agent into the animal and the occurrence of the first clinical signs of the disease (see www.woah.org/en/what-we-do/standards/codes-and-manuals/terrestrial-code-online-access/?id=169&L=1&htmlfile=glossaire.htm).

2.4.2.1 General properties

Influenza viruses are lipid-enveloped RNA viruses. Environmental persistence of AI viruses varies by subtype. In general, influenza viruses are fragile; however, some subtypes can persist longer under optimal conditions. The virus is stable over a pH range of 5.0–8.2.

2.4.2.2 Environment (including windborne spread)

Environmental conditions have a marked effect on AI virus viability outside the host. AI viruses are readily inactivated by heating and remain viable for longer periods in cold and humid environments (Beard et al 1984). They are sensitive to ultraviolet light; however, a long exposure period (>14 hours) is required for inactivation (Sutton et al 2013). Viability is prolonged in aerosols by high relative humidity and low temperature. Low temperature and high moisture levels also prolong virus viability in faeces.

Modelling found that theoretical persistence times of AI virus in leachates and water at landfill ranged from 23 to 696 days. Estimated persistence was longer at a pH of 6 at 4°C and in water in comparison to leachate (Graiver et al 2009).

Data from outbreaks of HPAI in poultry in south-eastern Australia suggest that outbreaks occur during periods of drought following a period of high rainfall (Klaassen et al 2011, Ferenczi et al 2016). Environmental conditions such as drought and flood affect waterbird abundance, regional distribution, and movement patterns and influence the number of serologically naive juveniles entering the population (Roshier et al 2002, 2008, Tracey et al 2004, Klaassen et al 2011, McEvoy et al 2015). For example, increased rainfall may lead to increased breeding season success for many wild bird species, more mixing of wild birds on water bodies, and increased movement as birds follow food and move into or out of flooded areas. Flooding may also lead to increased opportunities for wild birds to come into closer contact with poultry. In contrast, periods of drought may lead to increased wild bird densities in localised areas (such as around water sources). Therefore, it is likely that environmental conditions affect the abundance, distribution and movement of Anseriformes species and thus the apparent prevalence of AI viruses.

Hydrology variations (flood and drought) result in corresponding variations in wild bird long-range movement, breeding success, and local grouping at water sources. AI virus outbreaks are more likely to occur following high rainfall periods, hypothesised to peak 2 years after such an event (Ferenczi 2021).

2.4.2.2.1 Water

In laboratory inoculation studies, AI viruses appear most stable at a pH of 7.4–8.2, low temperatures (4–17 °C) and fresh-to-brackish salinities (salinity range of 0–20,000 ppm). Increasing water acidity, temperature or salinity shortens viability times for AI virus. However, there is significant variation between virus strains (Brown et al 2009). AI virus can be isolated from lake water, on-farm water and ice where waterfowl are present (Hinshaw et al 1979, Markwell & Shortridge 1982, Zhang et al. 2006). However, viral persistence in natural surface water in inoculation studies has been shown to be much lower than in distilled water assays (Nazir et al 2010). It is hypothesised that the presence of micro-organisms may antagonise viral viability as high microbial counts are negatively associated with persistence.

Estimated viral viability varies in studies utilising natural water depending on the experimental model (e.g. sandwich germ carriers vs suspension in surface water) and AI virus strain. Early viral suspension studies suggested that AI virus may remain infective in river water for up to 4 days at 22 °C and for more than 30 days at 4 °C (Webster et al 1978). Stallknecht et al (1990) predicted viral persistence in water at 1333 days at 4°C and 102 days at 28°C, although Brown et al (2007) estimated no HPAI viral persistence beyond 30 days at 28°C. Using a germ-carrier technique, Nazir et al (2010) estimated that AI virus may remain infective in lake water for up to 293 days at 10°C, 208 days at 0°C, 85 days at 10°C, 23 days at 20°C and 14 days at 30°C. However, there was significant variation in viral survival times across the strains included in the study. More recently, Zhang et al (2014) found that that virus remained viable in samples from water

bodies of important migratory bird habitats in China for up to 50 days at 4°C, 18 days at 16°C and 5 days at 20°C.

The water source is also important as town water is generally chlorinated and therefore a lower risk than inadequately sanitised surface/dam water that may be used as a water source for poultry.

2.4.2.2.2 Windborne and aerosol spread

Historically, windborne contamination has not been regarded as important in the spread of infection between properties. However, more recent research suggests that although windborne spread cannot fully explain farm-to-farm spread, it may have a role to play in some situations. During the outbreaks of HPAI in the USA in 2015, there was evidence of HPAI virus in air samples taken in the range of 70-150 m from infected facilities (Torremorell et al 2016). In the 2004 outbreak of HPAI H7N3 in British Columbia, Canada, viral genetic material was detected 800 m from an infected premises (IP) (Power 2005). In China, airborne AI virus was detected as far as 100 m downwind of a live poultry market (Wei et al 2018). Modelling conducted on windborne spread of HPAI between farms in the Netherlands 2003 outbreak concluded that the windborne dispersal of contaminated dust could contribute substantially to the spread of HPAI over short distances. The highest quantities of contaminated dust containing viable virus were detected 450m from an IP, with the quantity decreasing with distance. Modelling studies demonstrated that windborne spread may have accounted for up to 18-24% of total amount of transmission in the 2003 Netherlands outbreak, and over distances of up to 25km (Ssematimba 2012, Ympa et al 2013).

A study following the HPAI outbreaks in the USA in 2015 modelled air movement trajectories and virus concentration associated with 77 HPAI cases in Iowa. The results showed that the majority of infected farms may have received airborne virus, carried by fine particular matter, from infected farms within the same or neighbouring states. However, the modelled virus concentrations did not exceed the minimal infective doses for poultry (Zhao et al 2019).

In the 2024 New South Wales HPAI (H7N8) outbreak in the Hawkesbury Valley, aerosol spread up to 2.4km from the first infected premises (free-range and barn layer enterprise) may have resulted in transmission of the virus to other premises, including a broiler farm and several small backyard flocks. Wind strength and direction, overnight climatic conditions (mist with light winds) and absence of known horizontal contacts were consistent with the potential for aerosol transmission in this outbreak (Sharpe pers comm).

Evidence of aerosol transmission of swine influenza A (IA) virus by pigs under field conditions was shown with IA virus infectious aerosols being exhausted from pig barns and transported downwind, with viral RNA being collected in air samples 1.5km and, at significantly decreasing viral levels, at 2.1km from the barn (Corzo 2013).

Henzler et al (2003) reported that epidemiological investigations into an LPAI H7N2 outbreak in Pennsylvania suggested that depopulation of infected premises may have, in some situations, been responsible for spread of the virus to nearby farms. Disturbance of organic debris, including feathers, dust, and faecal matter, was observed as a result of depopulation activities, which may have contributed to spread of the virus.

While windborne spread of AI has not been definitively proven, available evidence suggests that it may play a role in spread of the virus. The distance that viable HPAI virus can be transported downwind in field conditions is unknown; however, windborne transmission should be considered as a potential mechanism of viral spread.

2.4.2.2.3 Soil-based substrates

Infection of SPF chickens with AI virus from contaminated soil-based substrates has been demonstrated under experimental conditions (Gutiérrez & Buchy 2012.) Estimates of the time in which AI virus persists

and remains viable in soil-based substrates range from 4 to 7 days (Gutiérrez & Buchy 2012, Horm et al., 2012, Poulson et al 2017). However, the ability of the virus to cause infection in natural systems such as lakes or streams due to contaminated soil-based substrate is unclear and is an area of ongoing research (Lang et al 2008, Perlas et al 2023).

Avian influenza viral RNA can be extracted from soil-based substrate including sediment, soil, sand, and mud (Densmore et al 2017, Gutiérrez & Buchy 2012, Horm et al 2012, Kuchinski et al 2024, Lang et al 2008, Nazir et al 2011, Perlas et al 2023, Poulson et al 2017). AI viral RNA is recoverable from the environment using molecular techniques for a longer period of time in soil-based substrates than infectious virus. Under experimental conditions using a germ carrier to promote survival of AI virus, AI viral RNA was found to be recoverable up to 394 days at 0 °C (with a range of 66–394 days) and 43–54 days at 10 °C⁴⁰ (Nazir et al 2011). AI virus RNA was able to be recovered from soil-based substrates in the environment over winter seasons and in below freezing conditions, however the infectivity of these samples was not assessed (Densmore et al 2017). Due to the ability to recover AI virus RNA from soil-based substrates Kuchinski et al 2024 proposed a whole genome sequencing method that may be used in surveillance of LPAI and HPAI viruses recovered from soil-based substrate. This technique was found to be able to accurately identify AI virus strains likely circulating in migrating wild birds by sampling the environment with the potential to reduce, cost and sampling bias in AI virus surveillance (Kuchinski et al 2024).

2.4.2.3 Susceptible species

See also Section 2.2.

2.4.2.3.1 Live domestic animals

2.4.2.3.1.1. Poultry

Experimentally, peak AI virus shedding occurs at 2–5 days post-infection, then rapidly declines. In rare cases, virus shedding has been prolonged; for example, with virus detection occurring for up to 72 days in turkeys. Virus shedding can be maintained for a longer period within a population of birds, and a domestic flock should be considered potentially infected unless elimination methods such as depopulation or effective vaccination programs have been implemented (Swayne, 2020).

During 2021–2022 H5Nx HPAI virus incursions in Europe, ‘silent’ infections in commercial broiler flocks occurred in 24 out of 230 (10.4%) of outbreaks, where commercial broilers did not show clinical signs and/or mortality. In these European outbreaks, turkeys and chickens (broilers) were the most affected populations (Gobbo et al 2022).

In an experimental study, Pantin-Jackwood et al (2007) reported that the pathogenicity of circulating Asian H5N1 HPAI viruses (various strains) in ducks varied depending on the virus strain and the age of the duck when infected and correlated with the level of viral replication in tissues. High titres of virus in organs, high viral shedding, and variable mortality enable ducks to circulate H5N1 HPAI viruses (Pantin-Jackwood et al 2007).

2.4.2.3.1.2. Aviary birds, including psittacines and canaries

AI viruses have been found in aviary birds (Easterday et al 1997), particularly psittacines (parrots, cockatoos and parakeets) and in other zoo birds (Usui 2020, FAO n.d.). Aviary birds have not been found to be the cause of infection in chickens or turkeys.

⁴⁰ Based on T(90) values where this is the time required for 90% loss of virus infectivity.

2.4.2.3.1.3. Game birds

AI virus was recovered from pheasants, partridges and guineafowl for up to 10 days after infection during the outbreak in the USA in 1983–84 (Swayne 2008). In experimental studies, infection has been detected in quail earlier than in chickens (Yee et al 2009).

2.4.2.3.1.4. Susceptible non-human mammals

2.4.2.3.1.4.1. Carnivores

While AI is principally an avian disease, AI viruses can also infect mammals⁴¹ especially carnivores. The noted increase in AI transmission to carnivores has been attributed to their consumption of infected birds, either through contaminated feed, carrion-eating or deliberate predation. One example of the infection pathway for carnivores is the 2006 fatal infection of a dog with H5N1 after it ate the carcass of an infected duck (Songserm et al 2006). Mammal-to-mammal transmission is assumed to have occurred in farmed mink (Agüero et al 2023). Studies have demonstrated that some mammalian species can excrete virus while remaining free of severe disease (Reperant et al 2008). Domestic cats infected with H5N1 display a range of clinical signs from subclinical through to fatality, with horizontal transmission between cats possible (Frymus et al 2021).

2.4.2.3.1.4.2. Dairy cattle

Indications are that the initial spread of the disease in dairy cattle originated from a single spillover event from wild birds in 2023, with continued disease transmission within the USA due to movement of livestock and fomites. As of September 2024, both genomic and epidemiological data analysis does not suggest the disease is spread onto dairy or poultry premises by migratory waterfowl.⁴²

2.4.2.3.1.4.3. Pigs

Pigs (domestic and feral) are a reservoir for mammalian influenza viruses and have been implicated in the generation of human pandemic viruses (Smith et al 2009). This is driven by the presence of both avian and mammalian-type sialic acid receptors (Nelli et al 2010). Cases of interspecies transmission identified between birds and swine included strains of H5N1, H9N2, H4N1, H4N6, H5N3, H10N5, H4N8, H6N6, H7N9 in China; H4N6, H6N2, H7N2 in the USA; H4N6, H3N3 in Canada; H7N2, H5N2 in South Korea; H5N1 in Nigeria and H5N1, H5N2, H9N2 in Egypt (Chauhan & Gordon, 2020). One of the established swine influenza lineages (Eurasian avian-like swine H1N1) is of avian origin (Pensaert et al 1981), demonstrating the capacity for AI to adapt and establish in swine.

Graaf et al (2024) found that nasal and alimentary experimental exposure of pigs to high pathogenicity avian influenza virus H5N1 clade 2.3.4.4b was associated with marginal viral replication without inducing any clinical manifestation or pathological changes. Only 1 of 8 pigs seroconverted, pointing to high resistance of pigs to clade 2.3.4.4b infection.

Kwon et al (2024) reported that experimental infection with mink-derived clade 2.3.4.4b, caused moderate to severe interstitial pneumonia with necrotising bronchiolitis with high titres of virus present in the lower respiratory tract of experimentally infected pigs and 100% seroconversion. Experimentally infected pigs shed limited amount of virus through the nasal and oral cavities, and importantly, there was no transmission to in-contact (sentinel) pigs. It was concluded that pigs are highly susceptible to infection with the mink-derived clade 2.3.4.4b H5N1 HPAI virus and may provide a favourable environment for HPAI viruses to acquire mammalian-like adaptations.

⁴¹ www.fao.org/animal-health/situation-updates/global-aiv-with-zoonotic-potential/bird-species-affected-by-h5nx-hpai/en

⁴² www.aphis.usda.gov/livestock-poultry-disease/avian/avian-influenza/hpai-detections/livestock

Arruda et al (2024) similarly assessed the susceptibility of swine to avian and mammalian HPAI H5N1 clade 2.3.4.4b strains using a pathogenesis and transmission model. All strains replicated in the lung of pigs and caused lesions consistent with influenza A infection. However, viral replication in the nasal cavity and transmission was only observed with mammalian isolates.

2.4.2.3.2 Live wild (including feral) animals

AI virus is infective for almost all wild waterfowl species, which are an important reservoir for the virus. In addition to HPAI H5Nx currently threatening wild and domestic birds across most of the globe, a virus that is highly pathogenic for poultry could emerge from the pool of viruses in wild birds at any time.

2.4.2.3.2.1. Waterfowl, seabirds and shorebirds

Anseriformes (waterfowl: ducks, swans, geese) and Charadriiformes (gulls, terns and shorebirds) are considered the main natural reservoirs for all avian influenza A viruses (Olsen et al 2006).

The pattern and duration of LPAI virus excretion varies between species (Costa et al 2011). A study by Latorre-Margalef et al (2009) demonstrated that the maximum duration of LPAI infection in migrating mallard ducks was on average 8.3 days with a mean minimum duration of virus shedding of 3.1 days. They demonstrated that the duration of LPAI virus shedding decreased during the 2002–2007 autumn migratory seasons, suggesting that mallard ducks acquire transient immunity for LPAI infection. The short virus shedding time also suggests that individual mallard ducks are less likely to spread the virus at a continental or intercontinental scale.

In a study of North American clade 2.3.4.4b H5N1 HPAI virus (Spackman et al 2023), all inoculated mallards shed the virus with the majority (65%) of birds shedding for up to 2 weeks post-challenge, which is considerably longer than what is normally found in ducks infected with other AI viruses. The highest quantities of AI virus shed, and highest proportion of ducks shedding, was on days 1–4 post-challenge.

2.4.2.3.2.2. Other wild birds

Current knowledge about virus shedding of avian influenza viruses in wild terrestrial birds is limited, however the pattern of virus shedding has been shown to be different among wild terrestrial birds, even in closely related species (Umar et al 2016).

Starlings have been shown to have limited intraspecies transmission, but they may pose a risk for spillover of AI viruses to poultry farms. They are unlikely to maintain infections without exposure to other species (Ellis et al 2021).

2.4.2.3.2.3. Wild and feral mammals

AI viruses can infect wild mammals, especially carnivores and marine mammals. As described in Section 2.4.2 (see ‘Carnivores’) infection of terrestrial carnivores is presumed to occur via consuming infected birds such as via predation, scavenging or the consumption of contaminated food/carcasses. Infection of marine mammals is thought to have occurred via close contact with, or ingestion of infected birds, or contact with marine environments contaminated with virus (Leguia et al 2023, Gamarra-Teledo et al 2023c, Elsmo et al 2023b, Elsmo et al 2023a).

Thousands of sea lions are reported to have died due to H5N1 in South America in 2023, the scale of which makes direct bird-to-mammal transmission unlikely to be the sole route of sea lion infection. There is speculation that mammal-to-mammal transmission may have occurred (Leguia et al 2023, Gamarra-Toledo et al 2023). Seals are not only susceptible to avian influenza infections but show evidence of adaptation of avian virus (H10N7) to a mammalian host (Bodewes et al 2016).

In a substantial proportion of mammalian cases, the PB2-627K mutation, which appears to increase viral replication rate in mammals, has been detected in the recovered viruses (Vreman et al 2023).

2.4.2.4 Nonsusceptible species

Since the emergence of HPAI virus clade 2.3.4.4b, the reported host range for these viruses has increased, with infection and clinical disease identified in a diverse range of species. Given the rapid advance in knowledge of susceptible species, it is difficult to definitively say that any bird or mammalian species is not susceptible. Even where an animal is not susceptible, it can still act as a fomite in transferring virus.

It has generally been concluded that mice and rats do not play significant roles in the spread of AI virus, but that insects may (Achenbach & Bowen 2011, Nielsen et al 2011). Rodents may act as vectors in the transmission of AI virus between wild birds and poultry, and between poultry farms, due to their abundance around farms, shared habitat with waterfowl, and ability to enter poultry sheds (Root & Shriner 2020).

Evidence regarding the susceptibility of rodents to AI infection and their role in transmission is varied. Shriner et al (2012) found that various LPAI subtypes (H3N6, H4N6, H6N2, and H4N8) can replicate in wild house mice and suggested a potential role for these rodents in the dynamics of AI outbreaks on poultry and gamebird farms (Root & Shriner 2020, Shriner et al 2012). More recent studies suggest rodents may be competent hosts for multiple AI subtypes. Li et al (2025) experimentally inoculated brown rats with H5Nx, H7N9, H9N2 and H10N8 subtypes. All strains infected the rats, inducing seroconversion without overt clinical signs; however, significant pathology in the respiratory tract was observed. The authors concluded that rats are permissive hosts for AI (Li et al 2025). Usui et al (2024) demonstrated viral replication in synanthropic wild rodents that were experimentally inoculated with H5N1 (clades 2.2 and 2.3.2.1). The authors concluded that wild rodents are susceptible to infection with avian-origin H5N1 subtypes and are replication-competent hosts (Usui et al 2024). The susceptibility of rodents to natural infection with AI virus and their role in AI ecology remains unclear. Since 2021, the FAO has listed a number of wild rodent species (family: Muridae) as affected by H5Nx HPAI.⁴³

2.4.2.5 Humans

Transmission from birds to humans can occur through handling of infected live or dead birds, or through close contact with:

- the faeces of infected birds
- respiratory secretions or saliva of infected birds
- uncooked infected birds, eggs or wild game bird products (via handling or consumption)
- contaminated products, such as litter
- contaminated dust and aerosols during destruction of buildings on infected premises.

The transmission of avian influenza virus from birds to humans is usually sporadic. People who are in close and repeated contact with infected birds or heavily contaminated environments are most at risk of acquiring avian influenza infection. However, due to ongoing circulation of various subtypes, outbreaks of avian influenza continue to be a global public health concern.⁴⁴

As of March 2025, there had been about 900 human infections with H5Nx, although the vast majority of those are due to H5N1 (Wille & Barr 2022). Since the emergence of LPAI virus H7N9 in China in March 2013, a total of 1567 laboratory-confirmed human cases have been reported, (case fatality rate of 39%). These reported cases included 40 instances of 2–3 human cases infected by a shared source (as of March 2019)

⁴³ <https://www.fao.org/animal-health/situation-updates/global-aiv-with-zoonotic-potential/bird-species-affected-by-h5nx-hpai/en>

⁴⁴ <https://www.woah.org/app/uploads/2025/07/25728-fao-woah-who-h5-assessment.pdf>

(WHO 2019a). Other virus subtypes found in poultry are also detected in humans, including H9N2 and H10Nx.⁴⁵ Of note is that some of the human cases that have been initially recorded as infections may have been due to carriage of the virus in the nose rather than a true infection. This is referred to as 'environmental contamination' which may result in positive nasal swabs without the presence of a true infection; this has been suggested in 2 recent cases in Spain (Aznar et al 2023).

AI viruses may be transmitted to pigs and potentially to humans. Reassortment of influenza A viruses may occur when pigs are concurrently infected with avian, porcine or human influenza viruses. Reassortment can lead to the emergence of new influenza viruses with avian or avian-like genes into the human population, potentially triggering a pandemic (Shortridge et al 1977).

There is serological evidence that H5N1 2.3.4.4b viruses infecting dairy cattle in the USA may also infect humans in contact with the cattle, although these infections are typically asymptomatic or with only mild symptoms (Garg et al 2025, Leonard 2025).

To date, the WHO has assessed that the global risk to public health from currently circulating H5N1 viruses as low, with a low to moderate risk for occupationally or frequently exposed persons, depending on the local epidemiological situation and the risk mitigation and hygiene measures in place.⁴⁶

2.4.2.6 Animal products

All animal products and byproducts from birds and flocks that are infected with AI virus, and farms or other sites contaminated with AI virus should be considered as high risk for the transfer of infection.⁴⁷

2.4.2.6.1 Meat, meat products, blood and casings

Viral titres in tissues destined for human consumption (predominantly skeletal muscle (meat) and liver) vary with virus strain, bird species, tissue type and clinical stage of infection.

The rate of detection of HPAI viruses in poultry meat appears to be higher during the early stages of infection (Beato & Capua 2011). Birds processed during the viraemic stage may contaminate other carcasses with blood or faecal material containing virus. Packaging and the drips that develop during storage are also important, because both can be contaminated with virus from infected carcasses.

Detectable titres of AI virus in chicken, turkey and duck blood have been reported. The viraemic phase of HPAI virus varies, depending on the virus strain and the species infected. Blood collected from infected birds (with or without clinical signs) may present some risk (Beato & Capua 2011). This pathway applies particularly to humans, for example during slaughter or processing of birds for consumption, but could also apply to any in-contact animals (Van Kerkhove et al 2011). Consumption of raw blood products is also considered a risk but unlikely to occur in Australia (Beato & Capua 2011; Van Kerkhove et al 2011).

Experimentally, HPAI virus has been found to remain infective in poultry meat for 160–210 days at 4°C (refrigeration) and 20–60 days at 20°C, with some variation between viral strain and between chicken, ducks and turkeys, with persistence being the longest in chicken meat (Beato et al 2012, Yaramoto et al 2017). Virus may be recovered from skeletal muscle of waterfowl that are not showing signs of disease. The rates of detection of HPAI viruses in poultry meat are higher during the early stages of infection, indicating an elevated risk linked to the trade of meat collected in the pre-clinical or early clinical phases of the

⁴⁵ <https://www.cdc.gov/flu/avianflu/reported-human-infections.htm>

⁴⁶ <https://cdn.who.int/media/docs/default-source/influenza/human-animal-interface-risk-assessments/influenza-at-the-human-animal-interface-summary-and-assessment--from-2-november-to-21-december-2023.pdf>

⁴⁷ Updated joint FAO/WHO/WOAH assessment of recent influenza A(H5N1) virus events in animals and people 25728-fao-woah-who-h5-assessment.pdf

disease (Beato & Capua 2011). Trials indicate, HPAI H5N1 virus titre in meat was high enough to be infective, and H5N1 can be detected in deep-frozen meat (Harder et al 2009, Cobb 2011).

In the liver, the survival time of HPAI virus is considerably shorter than in meat (20 days at 4°C and 3 days at 20°C; Yamamoto et al 2017).

Thorough cooking of meat (indicated by a change in colour of the meat from pink to white) results in no infective virus being recoverable (Swayne 2006). Improper cooking may allow AI virus to survive (Chmielewski & Swayne 2011); however, to date there is no direct evidence of AI transmission to humans via consumption of meat from an infected bird.

HPAI viruses have also been detected in the respiratory tract, gastrointestinal tract and bone marrow of experimentally infected chickens. Titres varied between studies and with subtype, but were 102.7–107.3 egg infectious dose (EID)₅₀/g in breast and thigh meat, 106.0 EID₅₀/g in lung tissue and 101.4–108.0 EID₅₀/mL in blood (Swayne & Beck 2005, Mase et al 2005, Muramoto et al 2006). The highest titres are found approximately 2–4 days post-infection. Refer to Appendix 2 for more information on infective dose and virus shedding.

2.4.2.6.2 Eggs and egg products

During systemic infection with AI virus, the virus replicates within the oviduct. Infected birds will stop laying (Sá e Silva et al 2013); however, both LPAI and HPAI viruses have been recovered from yolk and albumen, and HPAI virus has been recovered from the shell surface of chicken eggs (Cappucci et al 1985).

The most likely route of viral infection of eggs is faecal contamination of the external surface of the egg during passage through the cloaca or while in the nest. Vertical transmission may also play a role. Because AI virus can penetrate cracked or intact shells, unpasteurised or uncooked egg pulp and pulp products could also be a source of virus (Beato et al 2009).

Persistence during embryo development and incubation is most likely through shell contamination. However, AI viruses are lethal to embryos and hatching of experimentally infected eggs has not been achieved (Swayne & Halvorson 2008). Limited studies indicate that embryonated turkey eggs infected with influenza virus survived infection, and poults developed haemagglutination inhibition (HI) titres (Samadieh & Bankowski 1971). An experimental study demonstrated that incubation over a 2-day period at higher temperatures of 37 °C and 39 °C significantly increased LPAI virus growth (Lang et al 2011).

In naturally infected birds, H5N2 virus can be recovered from eggs (both albumen and yolk contents) laid by broiler breeders and commercial layers up to 18 days after the onset of clinical signs. AI virus can remain viable for up to 8 days in albumen and yolk of eggs stored at 10–18 °C. Virus recovery from albumen was higher than from yolk. Virus was also recovered, at a lower rate, in flocks not displaying clinical signs (Cappucci et al 1985).

The egg contents (albumen and allantoic fluid) and oviducts of naturally infected Japanese quail (*Coturnix japonica*) contained H5N1 virus titres of 104.6–106.2 EID₅₀/mL (Promkuntod et al 2006).

Commercial breeder duck eggs are typically laid on the floor, and are often heavily soiled with faeces, with many hens laying eggs in the same nest. External surface contamination with faeces is the major risk, but systemic circulating virus could also be shed into the internal contents during egg development in the oviduct.

2.4.2.6.3 Other

2.4.2.6.3.1. Edible birds' nests (EBN)

Several species of swiftlet build their nests from solidified saliva. These are a cultural delicacy for human consumption and are imported to Australia from Asia for this purpose. The product requires an import permit and must meet the conditions of retorted goods for human consumption.⁴⁸

2.4.2.6.3.2. Specialty duck products

There are a range of specialty duck products sold in Australia and used in the Asian food industry, including livers, feet, hearts, gizzards, intestine (casings), and specialty eggs. Most of these products are heat treated via cooking prior to consumption. The raw or cured products may pose a risk of human exposure to influenza viruses unless managed appropriately.

Another specialty item, duck blood is typically consumed cooked in soups and curries or as a clotted raw product (similar to beef tartare) in Asia; however, this is considered unlikely to occur in the Australian food industry.

Duck tongue is also a specialty and is normally sold as a pickled product that is usually fried before consumption.

Specialty duck egg products are also produced. This covers a range of salted egg yolks and cured 'century' eggs. Specialty duck embryos called 'baluts' are incubated for approximately 14 days and are sold in the intact eggshell for steaming or boiling before consumption.

2.4.2.7 Animal byproducts

2.4.2.7.1 Feathers

Feathers may be contaminated via faecal material or dust. Systemic infection has been demonstrated to localise in the feather follicle epithelium and AI virus has been detected in feathers from chickens, Japanese quail, turkeys, guineafowl, mallard ducks, call ducks (a bantam breed of domesticated duck raised primarily for decoration or as pets) and geese (Beato & Capua 2011).

H5N1 virus has been shown to replicate in swan feather epithelial cells, and could be detected on detached duck feathers for up to 160 days when the feathers were kept at 4 °C. When duck feathers were stored at 20 °C, virus remained infective for 15 days. Virus titres as high as 104.3 EID₅₀/mL were detected at 120 days (Yamamoto et al 2010). H5N1 virus survival times of 240 days were observed in cells from chicken feather pulp stored at 4 °C, and for 30 days at 20 °C (Yamamoto et al 2017).

A further study was conducted on chicken feathers infected with H5N1 by Yamamoto et al (2017) where the maximum periods for viral survival in feathers kept at 4°C was found to be 240 days and the viral infectivity at 20 °C was maintained for a maximum of 30 days.

2.4.2.7.2 Poultry meatmeal

Rendered meals produced from frames (boned-out skeletons), viscera, blood, feathers, feet, heads, necks, offcuts, and whole dead birds are added to poultry feed. Rendered meals may also be added to pet foods.

Heat-treated poultry offal meal and pet foods are usually cooked at temperatures above 100 °C for between several minutes and more than 1 hour, which is sufficient to inactivate AI virus (Swayne, 2006, Thomas et al 2008). However, not all pet foods and pet meat are heat-treated or pasteurised, and

⁴⁸ <https://bicon.agriculture.gov.au/BiconWeb4.0/>

uncooked pet foods and pet meats containing poultry products require treatment to kill AI viruses. If the procedure is not carried out properly or cooked product is subsequently contaminated by unprocessed product, AI virus could persist in the byproduct for several weeks (Beato et al 2009).

2.4.2.7.3 Waste products and effluent

Waste can be any of the unwanted byproducts of processing. Products that are used in the production of rendered meals may also be discarded as waste. In addition, there will be wastes from hatcheries, laboratories (cultures and specimens, including dead birds), farms and egg marketing establishments (unsaleable eggs, eggshells after pulping, solid egg fillers), as well as chicken manure and litter. AI virus can persist in these products and could be spread by vehicles that transport them, unless the products are treated before movement.

Few studies consider field viability of AI virus; however, one study stated that virus was isolated for up to 105 days following depopulation from wet manure (Fichtner 1987), while another found HPAI virus survived for 22 days in solid manure and 44 days in liquid manure (Utterback 1984). An investigation into a 2001-2002 LPAI H7N2 AI outbreak in broiler breeder flocks in the USA found that AI virus was not detected in manure, floor, dust or nest box swabs, 23 days after depopulation on the premises (Lu et al 2004).

Experimental studies have identified the importance of the combination of viral strain, temperature and relative humidity/moisture content on the persistence of AI virus, yet a thorough understanding of the process remain lacking (Lu et al 2003).

HPAI AI viruses remain viable for longer at cold temperatures, surviving in poultry faeces for as long as 35 days (H5N2) to 55 days (H5N1) at 4 °C (Beard et al 1984, Kurmi et al 2013), up to 13 days at 7°C (Wood et al 2010) and up to 5 days at 10°C (Stephens et al 2020). HPAI virus persistence in poultry faeces and litter declines with increasing temperature; surviving for up to 5 days at temperatures between 20-25°C, up to 24 hours at 32.2-37°C (Kurmi et al 2013, Stephens et al 2020) and up to 18 hours in poultry faeces at 42°C (Kurmi et al 2013).

HPAI viruses can remain viable in both wet and dry poultry faeces (Beard et al 1984, Wood et al 2010, Kurmi et al 2013) and high and low moisture poultry litter (Stephens et al 2020), with virus surviving longer in moist environments.

UV light influences viability. HPAI H5N1 virus in chicken faeces was inactivated within 4 days in shade at 25°C to 32°C and within 30 minutes in sunlight at 32°C to 35°C (Songserm et al 2006).

Manure handling can cause windborne spread of dust (Hauck et al 2017). Poultry flocks generate high concentrations of dust, but evidence for transmission of HPAI virus by dust is sparse (Spekreijse et al 2013). Windborne spread is further covered in Section 2.4.2.2.

2.4.2.8 Carcasses

Carcasses of wild or improperly disposed of birds and other animals infected with HPAI virus may pose an ongoing risk of transmission via consumption by carnivores (predators or scavengers) or via contamination of the environment (Reperant et al 2008; Liang et al 2023, Stallknecht & Brown 2009, Globig et al 2018)). H5N1 has been isolated from the carcasses of wild birds in an advanced state of autolysis (Ellis et al 2009).

2.4.2.9 Crops, grains, hay, silage, mixed feeds and bedding materials

These commodities may act as fomites for transmission of AI virus. Heat treatment during the manufacture of commercially prepared pellet poultry feed is usually sufficient to destroy AI virus. Uncovered storage of bedding straw that became contaminated with wild bird faeces has been implicated in transmission of H5Nx to commercial poultry in the United Kingdom in recent outbreaks.

2.4.2.10 Vehicles, including empty livestock transport vehicles

Vehicles, including empty livestock transport vehicles, can play an important role in transfer of AI virus from farm to farm. This is especially the case for vehicles involved in transporting dead infected birds, if there is inadequate containment and decontamination between premises (Bowes et al 2004).

Before the initial case is identified, it is possible that vehicles will play a role in disease spread between premises if routine biosecurity controls are not in place. Following confirmation of AI on a premises, biosecurity controls will be more stringent (see Section 6.4.7), reducing the likelihood of vehicles contributing to disease spread.

2.4.2.11 Equipment, including personal items

Persistence of AI virus in faeces and respiratory secretions is important in the spread of the virus via fomites. The moisture content of these components influences survival and thus spread of the virus over a wide area on footwear, clothing, equipment and other fomites. This is considered to be the main route of transmission of infection between premises.

Experience has shown that AI virus can spread very rapidly and over long distances by movement of contaminated materials such as bird cages, pallets, egg filler flats, manure, feed, clothing, equipment and vehicles. The time the virus remains viable on eggs and fillers is sufficient to allow wide dissemination. Overall, access of immunologically naive birds to fomites contaminated with infected faecal material poses the greatest risk of spreading infection.

High surface titres (around 10⁵ tissue culture infective dose (TCID)₅₀/mL) are required for transfer of human influenza viruses via fomites. At these high titres, virus survived on hard, nonporous surfaces (steel and plastic) at 28 °C and 35–40% humidity for 24–48 hours, and on cloth, paper and tissues for less than 8–12 hours. One study showed that virus could transfer from steel surfaces to hands for 24 hours, and from paper tissues to hands for 15 minutes. The authors concluded that a human shedding large quantities of influenza virus could transmit infection via a steel surface for 2–8 hours, and for a few minutes via paper tissues (Bean 1982).

2.4.2.12 Arthropod vectors

The poultry red mite, a common ectoparasite of poultry worldwide, has been identified as a potential mechanical vector in the spread of AI virus. In a study in 2016, mites ingested AI virus while feeding on infected chickens and then transmitted the virus to SPF chickens (Sommer et al 2016). Mechanical transmission by invertebrate or vertebrate vectors through contact with infected faeces is possible, but such transmission would be infrequent (Sawabe et al 2009).

Flying arthropods are also potential mechanical vectors. Mosquitoes collected at poultry farms during an outbreak of HPAI (H5N1) in central Thailand in 2005 tested positive for the virus (Barbazan et al 2008). A study has also demonstrated that H5N1 virus remained in the gastrointestinal tracts of house flies for at least 24 hours post-exposure (Tyasasmaya et al 2016). Feeding flies LPAI and HPAI viral suspension/solution resulted in positive virus isolations from these flies suggesting that flies are a possible pathway to consider for localised (i.e. within shed/farm) AI spread (Sawabe et al 2009, Nielsen et al 2011).

Darkling beetles can act as reservoirs and vectors of disease for a number of important poultry disease agents including avian influenza.⁴⁹

⁴⁹ www.clemson.edu/extension/camm/manuals/common_chapters/pch10c_03.pdf

2.5 Diagnostic criteria

2.5.1 Clinical signs

The clinical signs of AI virus infection are variable and influenced by the virulence of the virus, the species infected, the age of the infected individual, concurrent infection with other agents (e.g. infectious bronchitis virus, infectious laryngotracheitis virus), vaccination status, acquired immunity and environmental factors such as temperature. Pathogenicity in poultry can vary during an outbreak. Some birds may remain sub-clinically infected.

Clinical signs are commonly due to damage associated with:

- intracellular viral replication in tissues
- indirect effects of cell cytokine production (e.g. cytokine storm)
- ischaemia from vascular thrombosis
- coagulopathy or disseminated intravascular coagulation.

2.5.1.1 Infection with HPAI viruses

Clinical signs of infection with HPAI viruses result from replication of the virus in the respiratory tract, and subsequent systemic replication in the visceral organs and brain.

Whether the animal becomes infected depends on factors affecting exposure to the virus in the environment and individual or species susceptibility. The development of clinical signs depends on the interaction between the host and the virus, which is influenced by species or individual and virus strain factors.

Likelihood of exposure is greater in longer lived birds simply due to the opportunity for repeated virus introductions over time. However, the likelihood is also increased with greater environmental HPAI virus load in the region and high volume of traffic onto the farm, thereby highlighting the importance of biosecurity (Bertran et al 2016).

Clinical signs produced by infection with H5Nx clade 2.3.4.4 vary between species of bird; however, one paper reports a consistent finding in outbreaks across broilers, layer hens and Pekin ducks of an increase in mortality ratio that preceded (by 1–2 days), or coincided with, the observation of clinical signs. The suggested threshold for significance from this report was a daily mortality greater than 2.9 times the average weekly mortality, sustained for 2 consecutive days. Monitoring of the mortality ratio could provide an objective early indicator of virulent HPAI infection at a given chicken or duck property (Schreuder et al 2021).

2.5.1.1.1 Chickens and turkeys

In peracute cases involving sudden death, clinical signs may not be seen. Mortalities occur as early as 24 hours after the first signs of the disease and frequently within 48 hours. Mortality rates of nearly 100% have been reported for peracute and acute cases. In other cases, more diverse visible signs occur, and mortalities can be delayed for as long as a week.

Clinical signs in chickens and turkeys may include severe respiratory signs with excessively watery eyes and sinusitis; cyanosis of the combs, wattle and shanks; oedema of the head; ruffled feathers; listlessness/apathy; loss of appetite; diarrhoea; nervous signs; and sudden death.

A decrease in water and feed consumption may be noticeable at the flock/ farm level, and eggs from the birds may be misshapen or may be fragile. Up to 100% drop in egg production may be observed (Hassan &

Abdul-Careem 2020). The last eggs laid after the onset of illness frequently have no shells. Some severely affected hens may recover, but they rarely come back into lay.

Disease may spread very slowly in birds reared in cages, such as chickens and quail. Infection may be seen in a restricted area of a house or in single birds, but will slowly spread to adjacent cages (Capua et al 2000, Savill et al 2008).

The disease in turkeys is similar to that in chickens, but is often complicated by secondary infections such as fowl cholera, turkey coryza and colibacillosis.

2.5.1.1.2 Ducks (domestic)

H5N8 HPAI virus of clade 2.3.4.4b was noted in Europe to produce different clinical signs in ducks than in chickens, with ducks showing predominantly neurological signs. Along with neurological signs, decreased activity/ lethargy is a common finding (Schreuder et al 2021). Many ducks, including experimentally infected ducks, do not show any clinical signs at all. Geese also generally survive experimental exposure to H5N2 and H5N8 albeit with clinical signs that are generally more severe than in Pekin ducks (Grund et al 2018, Pantin-Jackwood et al 2017). There are also differences in the proportions of experimentally infected ducks showing clinical signs depending on the HPAI strain used and breed of duck, with the Muscovy being more susceptible than Pekin (Grund et al 2018). Experimentally infected Pekin ducks display disorders of the central nervous system such as tremor and opisthotonos (Grund et al 2018), torticollis, tremors of head, ataxia, and convulsions (Foret-Lucas et al 2023), as well as conjunctivitis and diarrhoea (Pantin-Jackwood et al 2017). Unlike chickens, Pekin ducks infected with HPAI H5N8 do not show signs of respiratory distress. The difference in clinical signs between the species may be related to the predominant site of viral replication – it is possible that slow viral replication in the lung provides enough time for viral replication in the brain to produce the neurological signs typical in ducks, whereas in chickens, intense viral replication in the lung underlies their clinical respiratory distress (Foret-Lucas et al 2023).

2.5.1.1.3 Ratites (commercially produced)

In studies on young ostrich poults, HPAI (H7N1) virus caused anorexia, depression, enteric signs (brilliant green urine, urates, severe haemorrhagic enteritis, haemorrhagic faeces), swollen throat and neck, nervous signs (including incoordination, wing paralysis, and head and neck tremors) and 30% mortality (Capua et al 2000, Mutinelli et al 2003). An HPAI (H5N2) virus obtained from an emu was inoculated intranasally and intratracheally into ostriches with virus subsequently isolated from multiple internal organs, and from cloacal and tracheal swabs for 2–12 days post-infection. The emu-origin virus did not cause clinical signs in the ostriches despite the widespread replication (Clavijo et al 2001). Elsayed et al (2022) found mortalities up to 90% in 60-day-old ostriches infected with HPAI H5N8, with signs of depression, loss of appetite, reduced production, oculo-nasal discharges and bleeding from natural orifices.

2.5.1.1.4 Wild birds

HPAI H5 virus strains (including 2.3.4.4b strains) have caused significant mortality events in a variety of wild bird species overseas. Wild birds infected with HPAI may show a range of clinical signs, from being non-clinical to presenting with neurological signs (e.g. ataxia, paralysis, seizures, tremors, abnormal posture), respiratory signs (e.g. conjunctivitis, increased nasal secretions, oedema of the head, dyspnoea), gastrointestinal signs (e.g. diarrhoea) or sudden death, similar to the signs seen in infected poultry (Knone et al 2018, Rijks et al 2022, Ailkie et al 2022, Gamarra-Toledo et al 2023, Nemeth et al 2023, Leguia et al 2023, Stallknecht et al 2007). Some clinical signs may be exacerbated by stress, such as during capture or handling.

Many wild ducks do not demonstrate clinical signs (van den Brand et al 2018, Poen et al 2018). However, the lack of clinical signs in wild ducks may be associated with cross-immunity provided by previous LPAI or HPAI H5 infections, as captive-bred wild mallards have been found to be highly susceptible to infection with

H5N8 clade 2.3.4.4b (Caliendo et al 2022b, Tarasiuk et al 2023). HPAI H5N1 is highly neurotropic in ducks and geese (Caliendo et al 2022a), so clinical signs most often include rhythmic dilation and constriction of the pupils, severe weakness, incoordination, tremors, and seizures. Studies on pre-2.3.4.4 lineages of HPAI H5 have also described cloudy eyes and ruffled feathers (Brown et al 2006).

For further information on clinical signs in wild birds, see the Wildlife Health Australia fact sheet *Avian influenza in wildlife in Australia*.⁵⁰

2.5.1.1.5 Mammals

Wild mammals infected with HPAI may show a range of clinical signs including neurological signs (ataxia, paralysis, seizures or tremors), respiratory signs (including increased nasal secretions, dyspnoea, tachypnoea), abortion or sudden death (including the potential for mass mortality events) (Gamarra-Toledo et al 2023, Leguia et al 2023, Puryear et al 2023, Elsmo et al 2023, Rijks et al 2021).

The USDA⁵¹ reports that common clinical signs in dairy cattle include decreased feed consumption, reduced milk production and thickened or discoloured milk. They reported that affected cows recover with supportive care, with little to no associated mortality.

2.5.1.2 Infection with LPAI viruses

2.5.1.2.1 Chickens and turkeys

Clinical signs of infection with LPAI viruses in chickens and turkeys range from inapparent to mild or severe respiratory disease. The signs can be confused with other respiratory tract infections.

Layer and broiler breeder chickens may show loss of appetite, decreased water consumption, depression, drops in egg production by up to 45% and misshapen eggs. Recovery of egg production typically occurs in 2–4 weeks; however, it should be noted that losses from reduced egg production may not recover to pre infection levels.

Generally, mortality is less than 5% in most avian species, unless exacerbated by secondary pathogens (Swayne et al 2013). Co-infections of the H9N2 virus with other respiratory pathogens, were reported to cause mortality up to 65% in some species (Agha et al 2023 and Haji-Abdolvahab et al 2019). The severity of infection in turkeys varies with the virus subtype, the age of the birds and the presence of secondary infections. Common signs include respiratory distress, rales and snicking, progressing to severe dyspnoea. Signs may also include swelling of the infraorbital sinuses, conjunctivitis, anorexia, fever, ruffled feathers and depression. Egg production may drop by 30–80%, with misshapen, fragile and whitish eggs produced during the acute phase. Mortality rates vary from 5% to 97%, depending on age, and morbidity may be up to 100% (Mutinelli et al 2003). Turkeys are generally more severely affected by reproductive disease than chickens (USAHA 2008).

2.5.1.2.2 Ratites (commercially produced)

In ostriches, clinical signs are highly variable and may be absent (with only serological evidence of infection) (Panigrahy & Senne 2003), but may include green urine, respiratory distress, enteritis, weakness and death. Clinical signs are more severe in young poults (Allwright et al 1993), in which mortality may be as high as 30% (Jorgensen et al 1998). Abolnik et al (2016) found that ostriches in southern Africa are frequently exposed (from wild birds) and become infected with both HPAI and LPAI and often show minimal clinical signs under good management conditions. In that study, it was found that clinical signs in H7-affected

⁵⁰ https://wildlifehealthaustralia.com.au/Portals/0/ResourceCentre/FactSheets/Avian/Avian_influenza_in_wildlife_in_Australia.pdf

⁵¹ www.avma.org/resources-tools/animal-health-and-welfare/animal-health/avian-influenza/avian-influenza-virus-type-h5n1-us-dairy-cattle

ostrich farms were more prevalent in chicks which presented with poor appetite, unthriftiness and mild bilirubinuria.

Respiratory signs were reported in rheas and emus (in the USA) infected with LPAI H5N2 and H7N1 viruses (Panigrahy et al 1995). No clinical signs were detected in infected emus during the 1997 outbreak in New South Wales (OCVO 2010). In the 2020 Victorian outbreak, young emus up to 7 weeks of age presented with lachrymation, swollen eyelids, sneezing, coughing and an increase in mortalities. Infection was confined to hatchlings/chicks and one-year old birds, with no active infection detected in older birds. Whole genome analysis confirmed that the virus detected belonged to an Australian H7 lineage avian influenza virus, with the most closely related LPAI H7 viruses detected in 2020 in wild birds.

2.5.1.2.3 Guineafowl, pheasant, partridge and quail

Natural LPAI virus infections in game fowl can be asymptomatic or symptomatic, with a range of clinical signs observed (Bertran et al 2014). Severe conjunctivitis was consistently detected in guinea fowl breeders infected with LPAI (H7N1) virus (Mutinelli et al 2003). Signs in guinea broilers included respiratory signs, nervous signs (including opisthotonus), torticollis and paralysis of the wings; mortality was up to 30%.

Pheasant and partridge are susceptible to infection and clinical disease. Some studies suggest that pheasant are more susceptible to LPAI viruses than chickens (Bertran et al 2014).

Quail may show no clinical signs of infection. Infection appears to spread slowly in quail, and they do not always produce an antibody response to infection. Therefore, surveillance programs using serology and virus isolation may require higher levels of testing than for chickens to detect infection.

2.5.1.2.4 Ducks

Infection with LPAI virus in domestic ducks and mallards typically results in subclinical infection; however, tufted ducks (wild) have been reported to present with neurologic signs, including head tilt, circling, loss of balance and drooping wings (Gavier-Widen et al 2012).

2.5.1.2.5 Other wild birds

Wild birds infected with LPAI viruses usually show no clinical signs.

2.5.2 Pathology

2.5.2.1 Gross lesions

In LPAI virus infections, lesions may be seen in the sinuses, characterised by catarrhal, serofibrinous, mucopurulent or caseous inflammation. The tracheal mucosa may be oedematous, with an exudate varying from serous to caseous. The air sacs may be thickened and have a fibrinous to caseous exudate. Catarrhal to fibrinous peritonitis and egg yolk peritonitis may be seen. Catarrhal to fibrinous enteritis may be seen in the caecum and/or intestine, particularly in turkeys. Exudates may be seen in the oviducts of laying birds (Easterday et al 1997).

In HPAI virus infections, including the peracute form of the disease, chickens may not show any gross lesions, dying 1–2 days post-infection. In Hong Kong and Italy in 1997, following acute infections in chickens, severe lung congestion, haemorrhage and oedema were observed in dead chickens; other organs and tissues appeared normal.

With the acute form of HPAI virus infection, more diverse visible lesions are evident, beginning 3–5 days after infection. Chickens have ruffled feathers, congestion and/or cyanosis of the comb and wattles, and swollen heads. The changes in the comb and wattles progress to dark red, then to blue, depressed areas of ischaemic necrosis. Internally, acute infections with HPAI viruses cause haemorrhagic, necrotic, congestive and transudative changes. The oviducts and intestines often have severe haemorrhagic changes. As the disease progresses, the pancreas, liver, spleen, kidney and lungs can display yellowish necrotic foci. Pancreatitis was a common lesion in chickens and turkeys infected with HPAI (H7N1) virus (Mutinelli et al 2003).

Petechiae and ecchymoses cover the abdominal fat, serosal surfaces and peritoneum. The peritoneal cavity is frequently filled with yolk from ruptured ova, associated with severe inflammation of the air sacs and peritoneum in birds that survive 7–10 days. Haemorrhages may be present in the proventriculus, particularly at the junction with the gizzard (Swayne & Suarez 2000).

In ostriches, gross lesions include severe haemorrhagic enteritis, and liver degeneration and necrosis (Capua et al 2000a, Mutinelli et al 2003).

Studies in wild bird species have indicated a variety of gross lesions may or may not be present, such as respiratory (pulmonary congestion, haemorrhage and oedema), pancreatic (necrosis, mottling), hepatic (necrosis), splenic (enlargement, necrosis), renal and cardiac (haemorrhage, myocarditis) abnormalities. (Kim et al 2015, Shearn-Bochsler et al 2019, Artois et al 2009, Lean et al 2022, Bröjer et al 2009). A recent study of wild common buzzards in the Netherlands reported only a small proportion of birds presenting with gross abnormalities, which included cardiac, brain and liver lesions (Caliendo et al 2022c).

In a recent study of HPAI infected mammals in the USA, the most commonly observed lesions were in the lungs (congestion, oedema, failure to collapse and haemorrhage). Other lesions were found in the brain, liver and kidney. Infrequent cardiac and gastrointestinal lesions were also noted (Elsmo et al 2023).

2.5.2.2 Microscopic lesions

The histological lesions associated with the gross pathological changes described above are not definitive for HPAI, although vasculitis in the brain and other organs may be highly suggestive of the disease.

2.5.2.3 Pathogenesis

2.5.2.3.1 Gallinaceous birds

Pathogenesis begins with inhalation or ingestion of infectious AI virions. In both HPAI and LPAI virus infections, the initial replication site is usually the nasal epithelium. The viral HA adsorbs to host endothelial cell receptors containing sialic acid-bound glycoproteins. Proteolytic cleavage of the HA into H1 and H2 is essential for fusion and initiating receptor-mediated endocytosis. For all AI pathotypes, trypsin-like enzymes on the surface of epithelial cells of the respiratory and intestinal tract allow cleavage of the surface HA and entry of virus particles into the cell, where multiple replication cycles occur with the release of infectious virions.

For LPAI viruses, replication is limited to tissues with trypsin-like enzymes – usually the mucosal tissues of the respiratory and intestinal tracts – but may spread systemically in some species to kidney tubules, pancreatic acinar epithelium, oviduct and other epithelial cells that have trypsin-like enzymes (Swayne & Suarez 2000).

HPAI viruses can be cleaved by protease (furin-like) enzymes in addition to trypsin-like enzymes; this allows them to enter and replicate in other tissues. After initial replication in the respiratory or intestinal epithelium, virions may invade the submucosa, replicating in endothelial cells, and spread via the vascular or lymphatic system to infect other cell types in visceral organs, brain and skin. Alternatively, they may become systemic before extensive replication in vascular endothelial cells. Macrophages and heterophils play a key role in systemic virus spread (Swayne & Halvorson 2003). Initial visceral replication may be seen as early as 24 hours after intranasal infection, with high titres present by 48 hours.

2.5.2.3.2 Nongallinaceous birds

In nongallinaceous birds, the pathogenesis is less well understood.

In a study of ducks infected with different Asian-origin H5N1 AI viruses, viral pathogenesis was similar to that of chickens, except that in the case of ducks there is neither replication of virus in endothelium nor are there any associated lesions in the endothelium. Microscopic lesions and AI viral antigen were observed most frequently in the nasal cavity, brain, heart, adrenal glands, and pancreas (Pantin-Jackwood & Swayne 2007). An earlier study of A/chicken/Hong Kong/220/97 (H5N1) in a variety of species including finches, sparrows, starlings and budgerigars found that histologic lesions and the corresponding viral antigen were observed in multiple organs, especially in the nasal cavity, brain, pancreas, spleen, adrenal glands, and ovary, except for the starlings in which viral antigen was not observed in any collected tissues (Perkins & Swayne 2003).

2.5.3 Differential diagnosis

AI in birds can produce non-specific clinical signs and lesions and may be indistinguishable from other diseases based on clinical signs and postmortem morphological findings.

Differential diagnoses include the following (non-exhaustive list):

- viral diseases
 - Newcastle disease
 - infectious laryngotracheitis
 - avian paramyxovirus
- bacterial diseases
 - erysipelas
 - fowl cholera (acute pasteurellosis)
 - acute *Escherichia coli* infection and other septicaemias
 - mycoplasmosis
 - ornithobacteriosis
 - infectious coryza
 - avian chlamydiosis
- fungal diseases
 - aspergillosis
- other causes
 - acute poisoning
 - heat stress/exhaustion
 - physical exhaustion
 - severe water deprivation
 - misadventure associated with high mortality (e.g. smothering, heat stress)
 - botulism.

2.5.4 Laboratory tests

Because clinical and pathological changes are not definitive for AI, diagnosis must be confirmed by isolation of the virus or by characterisation of fragments of its genome (WOAH 2022). Relevant laboratory tests should be performed to exclude Newcastle disease and bacterial septicaemias from the differential diagnosis, particularly to identify mixed infections with less pathogenic forms of AI.

If an outbreak is confirmed to be caused by an HPAI virus, this agent may also be classified as a security sensitive biological agent (SSBA), to which regulatory requirements (e.g. for handling and reporting) may apply. This may be the case if there is proven infection of humans and there is an outbreak of human disease. For further information, see the **AUSVETPLAN Management manual: *Laboratory preparedness***.

2.5.4.1 Samples required

2.5.4.1.1 Birds

Samples should be taken from both live, clinically affected birds and recently dead birds.

Cloacal, oropharyngeal or tracheal swabs placed in viral transport media (VTM) and/or fresh faeces; and from live birds clotted blood/serum may be collected.

Unpreserved alimentary tract tissues (proventriculus, pancreas, intestine, caecal tonsil); respiratory tract tissues (trachea, lung); cloacal, oropharyngeal and tracheal swabs; and fresh faeces should be collected from recently dead or euthanised birds.

2.5.4.1.2 Non-human mammals

Where indicated, samples should be taken both from live, clinically affected and recently dead non-human mammals.

Upper respiratory tract (URT) swabs (e.g. nasal or tracheal/oropharyngeal) and/or samples from the lower respiratory tract (LRT) (e.g. bronchoalveolar lavage) may be collected.

Unpreserved LRT/lung and central nervous system tissue can be collected from recently dead or euthanased mammals.

2.5.4.2 Transport of specimens

Specimens should be submitted in accordance with agreed state or territory protocols. Specimens should initially be forwarded to the state or territory laboratory for appropriate analysis and assessment of whether further analysis will be required by the CSIRO Australian Centre for Disease Preparedness (CSIRO-ACDP), Geelong.

If the state or territory laboratory deems it necessary, duplicate samples of the specimens should be forwarded to CSIRO-ACDP for emergency disease testing, after the necessary clearance has been obtained from the chief veterinary officer (CVO) of the state or territory of the suspect case, and after the CVOs of Victoria and Australia have been informed about the case and the transport of the specimens to Geelong (for the first case). Sample packaging and consignment for delivery to CSIRO-ACDP should be coordinated by the relevant state or territory laboratory.

For further information, see the **AUSVETPLAN Management manual: *Laboratory preparedness***.

2.5.4.3 Packing specimens for transport

Unpreserved tissue, swab and blood specimens should be forwarded with water ice or frozen gel packs (dry ice or liquid nitrogen if a delay of more than 48 hours is expected) in an International Air Transport Association–approved specimen transport container.

For further information, see the **AUSVETPLAN Management manual: *Laboratory preparedness***.

2.5.5 Laboratory diagnosis

The initial approach to AI diagnosis is screening by real-time PCR. Primary screening uses a pan-influenza A assay, as well as specific H5 and H7 assays either concurrently or on samples giving positive results in the pan-influenza A assay. Further subtype-specific assays may also be run, if required. Any positive samples are further characterised by molecular analysis and culture in eggs for in vivo pathotyping, as well as molecular and antigenic characterisation on isolates. Analysis of viral genetic sequence data allows assessment of pathogenicity (see ‘Agent characterisation’, below), as well as more-detailed phylogenetic analysis.

Isolates obtained from egg culture are identified antigenically by hemagglutination inhibition (HI), as well as with molecular tools.

2.5.5.1 CSIRO-ACDP tests

The testing method used by CSIRO-ACDP is shown in Figure 2.1. Further details of tests currently available at CSIRO-ACDP, some of which are supported through the Laboratories for Emergency Animal Disease Diagnosis and Response (LEADDR) network, are shown in Table 2.3.

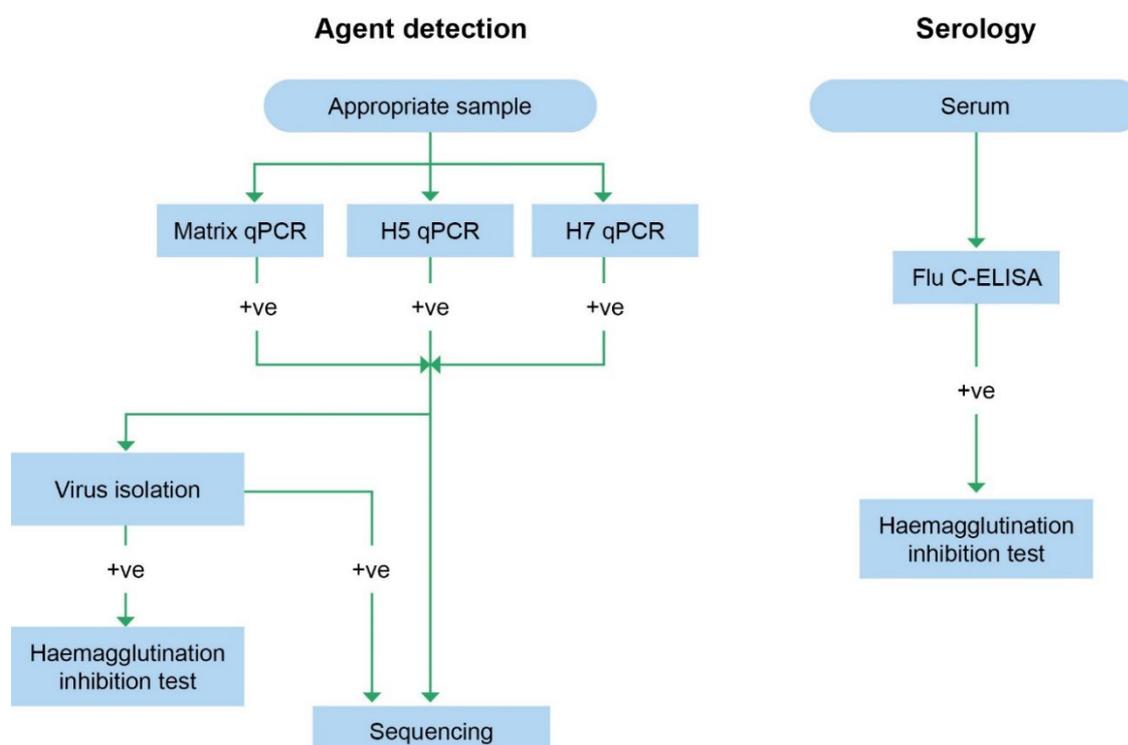


Figure 2.1 The current approach to diagnostic testing at CSIRO-ACDP

Table 2.3 Laboratory tests currently available at CSIRO-ACDP for the diagnosis of avian influenza

Test	Specimen required	Test detects	Time taken to obtain result
Agent detection			
Real-time PCR ^a	Swabs, tissues	Type A influenza, H5 and H7 subtypes, some LPAI subtypes	<1 day
Immunohistochemistry	Formalin-fixed tissues	Viral antigen	2 days
Virus isolation in embryonated eggs	Swabs, tissues	Virus	2–10 days
Agent characterisation			
PCR and sequencing	Swabs, tissues or virus isolate	Viral RNA	1–2 days
Antigenic subtyping (HI)	Virus isolate	Specific HA and NA antigens	1–4 days
Intravenous pathogenicity index	Virus isolate	Virulence of virus	2–10 days
Serology			
ELISA ^a	Serum	Antibody (influenza A)	1 day
HI	Serum	Antibody (specific HA serotypes)	1 day

ELISA = enzyme-linked immunosorbent assay; HA = haemagglutinin; HI = haemagglutination inhibition; LPAI = low pathogenicity avian influenza; NA = neuraminidase; PCR = polymerase chain reaction

^a Test also supported as part of the LEADDR network.

2.5.5.2 Other tests

Agent characterisation: tests for virus subtype

AI viruses are subtyped on the basis of the sequence of the HA and NA genes, as well as their HA and NA antigens. Details of tests for subtyping (PCR and sequencing, and antigenic subtyping) are in Table 2.3.

Agent characterisation

Tests for pathogenicity

The pathogenicity of an influenza virus isolated from a bird is generally assessed by sequencing the part of the gene encoding the cleavage site of the HA protein of the virus (molecular pathotyping). In some circumstances it is necessary to verify high or low pathogenicity of a virus isolate using an *in vivo* chicken pathogenicity test (intravenous pathogenicity index) (WOAH 2022).

For a timely diagnosis, molecular pathotyping is the preferred method of determining the pathogenicity of an AI virus in Australia. Once an outbreak virus has been characterised, virus detection and virus isolation are generally sufficient to confirm further virulent infections.

Tests for previous infection

Evidence of previous or recent AI virus infection can be obtained by testing serum for influenza A group-specific antibody using an ELISA, and by testing for subtype-specific antibody to the HA or NA antigens using an HI test or ELISA, respectively.

Use of serology for AI testing has several limitations:

- HI testing at a single point in time does not provide an indication of recent infection. Repeat blood samples collected 2–3 weeks apart may allow further interpretation of the HI results if a change in titre can be demonstrated
- Positive serology results can indicate that the bird had exposure to an antigenically similar virus, and not the specific virus present in the outbreak situation.

Positive serology, however, may be useful to assess the extent of subclinical infection on a farm before development of clinical disease.

Field tests

Currently, no field tests are approved for use during an EAD response involving AI in Australia.

2.6 Resistance and immunity

Host responses to AI virus may vary, mainly with the subtype and strain of the virus, and the species infected. Different subtypes and strains may stimulate different responses, and different species may develop different immune responses. Immunity has been most closely studied in domestic birds.

2.6.1 Innate immunity

Innate immunity is the first nonspecific line of defence against pathogens, comprising physical and chemical barriers to infection, and a range of antimicrobial mechanisms and factors at the cellular and humoral levels. Birds have well-developed innate immune systems. Chickens are generally less able to resist infection with HPAI virus than ducks, which are more efficient in clearing the virus through the capacity of their innate immune system (Barber et al 2010).

2.6.2 Acquired immunity

Maternal (passive) immunity (passed to progeny via the egg) has been demonstrated in both naturally infected chickens and vaccinated chickens. During natural infection, maternal antibody provided limited protection against HPAI (H5N1) virus (De Vriese et al 2010).

Ducklings are partially protected by maternal antibodies (IgY) passed through egg yolk. As in chickens, maternal antibody may interfere with development of immunity in vaccinated ducklings (Magor 2011).

2.6.2.1 Humoral immunity

Both infection with AI viruses and vaccination elicit a humoral antibody response at both systemic and mucosal levels. The mucosal antibody response (mainly IgA) has not been well characterised (Swayne & Halvorson 2008), but probably plays an important role in recovery from infection and protection from further infections, especially with LPAI virus.

The basis of protective humoral immunity is the development of neutralising antibody against the 2 major surface proteins, HA and NA (Swayne & Halvorson 2008). These antibodies do not cross-neutralise viruses of different HA or NA subtypes. The neutralising antibodies against the HA and the NA interfere with viral replication in different ways (Qiao et al 2003). In general, inactivated vaccines provide protection through humoral immunity.

Birds other than ducks (see below) that were infected with LPAI viruses were protected against challenge with virulent strains having similar surface antigens (Seo & Webster 2001).

In chickens, the IgM response is measurable as early as 5 days post-infection, and IgY can be detected shortly after this. Detectable antibody following infection with a virus of the same HA subtype persists for varying lengths of time, from 6 weeks to 12 months (Fichtner 2003, Pearson et al 2003). Antibody titres to different antigens in various bird species indicate that antibody production may be greatest for chickens, followed by (in decreasing order) pheasants, turkeys, quail and ducks (Suarez & Shultz-Cherry 2000).

In ducks, IgM is produced first (at 3–5 days), but is transient and quickly replaced by IgY (day 12) and IgA. IgA is an important mucosal surface secretory antibody in ducks, and bile IgA has virus-neutralising and HA-inhibition activity. Mucosal surfaces of the respiratory and intestinal tracts of ducklings are not protected by IgA during the first 2 weeks of life. IgY is the primary serum antibody in ducks (Magor 2011).

Ducks are usually found to mount a poor HA antibody response to natural and experimental AI infections compared with chickens, based on HI testing. Ducks may be reinfected with the same or similar influenza strains, and do not seem to mount a secondary immune response (Magor 2011). Assays for measuring antibody response typically use secondary antibodies specific for chicken IgY; they are therefore highly

species specific, and are less reliable for diagnosis in ducks, geese or other wild bird species (Magor 2011, Curran 2012).

2.6.2.2 Cellular immunity

Cellular immunity contributes minimally to protection against HPAI virus infection and disease in poultry, because mortality following HPAI is rapid, precluding the 1-week timeframe needed to induce a cytotoxic T-lymphocyte (CTL)–specific immune response (Swayne & Kapczynski 2008). However, some studies indicate that cellular immunity can limit the severity and duration of disease following HPAI virus infection in chickens and turkeys. Such immunity can be gained either by vaccination or by exposure to non-lethal infection with AI virus. Whereas inactivated vaccines do not stimulate memory T lymphocytes, live vaccines stimulate nucleoprotein-specific CTLs. This results in protection from lethal challenge, and allows more rapid clearance of virus and recovery following influenza virus challenge. There is also some evidence of cross-protection in chickens from cellular immunity following H9N2 infection against lethal H5N1 virus challenge (Seo & Webster 2001).

2.7 Vaccination

Effective vaccination reduces susceptibility to infection. When infection does occur, it reduces clinical signs of disease and the amount of virus shed into the environment. Under some circumstances, vaccination may be used alongside other control methods to support eradication programs. Vaccination of commercial flocks is being considered or actively recommended in some countries overseas as part of an integrated disease control approach where biosecurity measures alone have not been able to effectively control HPAI, such as in EU member states.⁵² It is also permitted for use in licensed zoo birds in the United Kingdom.⁵³

Human and animal influenza virus vaccines continue to be developed and assessed, in response to overseas incidents of human infections and deaths with H5 and H7 AI viruses. Although new technologies will influence poultry vaccines in the future, the types of vaccine currently licensed by overseas authorities for use in poultry include:

- inactivated whole AI virus vaccines
- live genetically modified vaccines, including turkey herpesvirus (HVT)-vectored
- fowl pox–vectored and Newcastle disease virus–vectored vaccines
- inactivated genetically modified vaccines
- RNA particle vaccine.

Many vaccines used around the world are inactivated whole AI virus (antigen) vaccines in an oil-based emulsion adjuvant, produced according to WOAHP standards. Inactivated vaccines are made by formalin treatment of infected allantoic fluids from chicken embryos to inactivate the agent(s), and adjuvanted by making a water-in-oil emulsion using mineral oil.⁵⁴

Immunity to inactivated whole AI virus vaccines results primarily from response to the HA protein and, to a lesser degree, the NA protein. These vaccine technologies produce safe, pure and potent vaccines; commercial inactivated vaccines have been shown to protect against disease and prevent mortality, particularly if the antigen in the vaccine is closely matched with the field virus. However, they require handling and injection of individual birds. Another significant disadvantage is that they are not able to prevent virus shedding in chickens challenged with antigenically different viruses. Viral shedding supports the persistence of the virus in the field. Thus, the vaccine strain used has to closely match the circulating field strains, which means that the vaccine may need to be frequently updated to account for antigenic drift of field strains.

The inactivated vaccines use homologous HA determinants (e.g. H5) and either a homologous or heterologous NA determinant to provide protection against known current field strains of AI virus. The use of heterologous NA subtype vaccines provides an opportunity to use serological surveillance to detect circulating field virus by detecting antibodies to the NA subtype of the field virus – that is, a DIVA (differentiating infected from vaccinated animals) strategy. Vaccine technologies are being developed that will enable DIVA testing based on viral proteins other than the NA, removing the need to use heterologous NA subtypes for DIVA purposes.

Live fowl pox–and HVT–vectored AI virus vaccines have the advantages that the subsequent immunity is not suppressed by maternal antibodies (Bublout et al 2006), and they stimulate both cellular and humoral antibody responses. The vaccines can also be administered by injection at 1 day of age in the hatchery. A

⁵² www.woah.org/en/avian-influenza-vaccination-why-it-should-not-be-a-barrier-to-safe-trade/

⁵³ www.gov.uk/government/publications/avian-influenza-bird-flu-vaccination/avian-influenza-bird-flu-vaccination

⁵⁴ Following vaccination of hens with inactivated vaccine (H5N1/H5N2), progeny demonstrated high to moderate levels of maternal antibody at 1–5 days of age and low levels by 7 days. Progeny of vaccinated hens that were vaccinated at 5 days of age had a poorer response to vaccination (and therefore increased susceptibility to AI virus infection) than those vaccinated at 10 days, because of interference from maternal antibody (Maas et al 2011).

Newcastle disease virus–vectored AI vaccine has recently been developed that can be administered by spray in the hatchery.

Possible problems with the use of vaccines include the following (EFSA 2023):

- low vaccine efficiency, which fails to fully protect vaccinated birds and prevent new outbreaks
- vaccine failure due to host-specific factors that could reduce immune response of vaccinated birds
- inadequate vaccine coverage to stop virus circulation
- inefficient surveillance that may lead to the inability to detect field virus in vaccinated flocks, resulting in clinically silent circulation of HPAI virus which puts nonvaccinated birds (such as wild birds) at risk
- inactivated vaccines have been associated with antigenic drift in field viruses
- vaccination may favour the emergence of more virulent variants through indirect selection of viruses
- impractical vaccine administration (e.g. booster requirements, no mass-application)
- trade restrictions
- availability of effective vaccines for diverse poultry species.

Vaccines may decrease in efficacy when used for a long period, as a result of antigenic drift in field viruses, and should be updated to match the predominant antigenic types.

Vaccination for free-ranging wild birds is generally considered impractical with currently available vaccination strategies. Emergency vaccination of wild birds could be considered as an adjunct to other control measures for rare and valuable wild animals under specific situations (WOAH 2023) but has had very limited application worldwide to date. A vaccination program for intensively-managed, free-ranging endangered Californian condors was undertaken in the USA in 2023 after it was decided that HPAI mortalities in the population posed a threat to the survival of the species.⁵⁵ New Zealand has also undertaken a vaccine trial for some key threatened species in captive populations.⁵⁶ Vaccination has been used overseas to help protect some captive populations of wild species (Vergara-Alert 2011, Philippa 2007), such as those in zoos.

2.8 Treatment of infected animals

There is no effective treatment for AI virus infection, and prognosis for birds affected with virulent disease is generally poor. There are some limited reports of birds surviving infection. However, in general, those that do survive are in poor condition and may continue to shed virus, thus posing a risk to other birds, populations or locations, and the welfare of the affected birds may be compromised. Additionally, poultry may resume laying only after a period of several weeks, if at all.

Very limited application of supportive care for infected wildlife has been undertaken overseas. The potential use of antiviral drugs in birds has also undergone some research.

⁵⁵ www.fws.gov/program/california-condor-recovery/southwest-california-condor-flock-hpai-information-updates-2023#:~:text=All%20condors%20are%20vaccinated%20by,minimize%20the%20likelihood%20of%20mortality.

⁵⁶ www.doc.govt.nz/globalassets/documents/our-work/wildlife-health/hpai-bird-flu-vaccination-trial-general-factsheet-june-2024.pdf

3 Implications for Australia

3.1 Potential pathways of introduction or evolution

Strains of HPAI virus could be introduced into Australia or evolve in Australia through:

- migratory birds and other natural non-migratory movements of infected wildlife, including but not limited to wild birds
- illegal importation of contaminated goods
- importation of contaminated poultry products, fomites, inanimate objects or people
- evolution from domestically circulating LPAI viruses.

3.1.1 Migratory and non-migratory movements of birds and other wildlife

Long-distance migratory birds of the orders Charadriiformes (notably shorebirds) and Procellariiformes (notably shearwaters) move to Australia during the spring migratory period (August to November) each year.

Anseriformes (waterfowl) within the Australo-Papuan region are nomadic all year-round. The majority of these frequent yet irregular nomadic waterfowl movements are confined to locations east of the Wallace line and are driven by strong environmental conditions within the region (Ferenczi et al 2016, Purnell 2022), with no apparent or consistent seasonal patterns, in contrast to movements by long-distance migratory birds. The minimal waterfowl movement across the Wallace line has been thought to limit HPAI virus incursions from Southeast Asia to Australia (East et al 2008b). However, it should be noted that a small number of waterfowl, shorebird and sea bird species have distributions that extend across this line, such as the Pacific Black duck and Spotted Whistling duck (Purnell 2022; Roshier et al 2012) and significant temporal, spatial and research gaps preclude a full understanding of bird movements between Australia and the islands to the north (Purnell 2022).

Incursion via the Antarctic region is also plausible but with high uncertainty due to limited data available on population structures and fine-scale tracking.

Historically, the risk of HPAI incursion into Australia via natural movements of wild birds has been assessed as low, owing to the low likelihood that HPAI infected birds would survive long enough to enter via known migratory or movement routes. However, the risk has recently increased due to changes in a number of risk factors associated with current HPAI H5Nx clade 2.3.4.4b viruses which significantly differ from previous HPAI H5 viruses.⁵⁷ Risk factors include increased avian and mammalian host and geographic range, and the ability to spread via a wide range of avian species including both poultry and wild birds, in particular the ability of wild birds to directly infect poultry with HPAI viruses. The virus also exhibits increased pathogenicity and increased replication and viral shedding in wild birds, with an increased persistence in the environment. Hence the risk has increased due to factors influencing both the likelihood of entry and exposure of HPAI, as well as the consequence of HPAI incursions in Australia.

3.1.2 Evolution from domestic LPAI viruses

Evolution of high pathogenicity viruses from domestic H7 LPAI viruses circulating within commercial poultry is characteristic of all Australian HPAI outbreaks to date. Waterfowl or other wild birds can be non-clinical carriers of LPAI virus and can transfer LPAI virus to domestic poultry via close contact, directly through fomites or indirectly through contaminated drinking or cooling water. This is a constant risk and risk levels

⁵⁷ https://wildlifehealthaustralia.com.au/Portals/0/ResourceCentre/BiosecurityMgmt/HPAI_incursion_risk_assessment_Australia.pdf

may change over time based on factors such as season and distribution of both commercial poultry and wild bird species, and genetic characteristics of circulating viruses that influence their potential for mutation and adaptation.

3.2 Social, economic and environmental effects

One of the biggest impacts of an outbreak of HPAI or LPAI (H5/H7) involving the poultry industry would be on the domestic economy. Chicken meat and eggs are produced very efficiently in Australia and provide a relatively low-cost source of animal-based protein to the population, which is reflected in the ever-increasing consumption rate. Loss of availability of these products could cause economic stress to much of the population and the domestic economy.

Domestic supply may be affected when the disease is detected. The effects may be more severe if the disease is widespread, or a large production region is affected, and movement restrictions impede the delivery of product to markets.

For affected producers, the main financial losses during an outbreak would be from flock mortalities, which can be high, and reduced productivity of affected flocks. For contracted farmers and their workforce, there may also be a prolonged period of lost income while infected premises are depopulated and decontaminated during an outbreak response. A stamping-out or modified stamping-out policy would cause further loss of income to both affected properties and poultry companies for an extended period. Where compensation applies, it is paid to the owner of the birds; contracted growers who do not own the birds may therefore experience considerable financial loss.

Disruption to the flow of product, potential declines in human consumption of poultry and poultry products, and subsequent decreases in production may cause job losses on farms, and in service and associated industries (e.g. feed mills, transporters), depending on the time it takes to bring the outbreak under control. In large outbreaks, flow-on effects to whole communities can be expected to affect other industries, including tourism. Even a small outbreak would result in dislocation of the industry and its normal marketing patterns. Infection in grandparent and foundation flocks would cause loss of some valuable genetic material (see the **AUSVETPLAN Enterprise manual: Poultry industry** for information on the structure of the poultry industry).

Export markets for poultry products and genetic material are likely to close immediately upon declaration of an HPAI outbreak. The extent of closure will depend on the market in question.

Zoos and premises holding captive birds, including pet shops and aviaries, may be affected directly by the outbreak, or indirectly through movement controls, restricted public access or public concern around being in the presence of birds.

Strains of HPAI viruses circulating overseas have resulted in die-offs of waterbirds and deaths of multiple avian species, including birds of prey. During the 2016–17 epizootic of an HPAI (H5) virus (Guangdong lineage), 112 wild bird mass mortality events were reported across Europe (Alarcon et al 2018). During the same epizootic, approximately 13 600 birds of 71 species were reported dead in the Netherlands (Kleyheeg et al 2017). Between 2016 and 2018, marine birds made up the largest group of reported species mortalities during the HPAI (H5N8) epidemic in Africa (including South Africa); terns, especially swift terns, were the worst affected systematic group, followed by the African penguin (*Spheniscus demersus*) (Khomenko et al 2018). Since 2021, H5Nx clade 2.3.4.4b, has caused unprecedented outbreaks in wild birds and poultry in all continents except Oceania (including Australia and New Zealand). Between October 2021 and June 2023, there were 10,489 reported outbreaks (i.e. notifications to WOA) of HPAI in wild birds, resulting in at least 70,601 reported wild bird deaths. Given these reports only represent detected and officially reported mortalities, the actual scale of wild bird mortalities is likely to be in the millions (Klaassen

& Willie 2023). Clade 2.3.4.4b has also resulted in unprecedented morbidity and mortality events in terrestrial and aquatic mammals.

Sites of wild bird die-offs during HPAI epidemics have included wetlands of international importance (Ramsar sites) for waterbirds in west Africa, Europe and Asia. H5Nx clade 2.3.4.4b outbreaks in wild bird species of high conservation status have had negative impacts on decades of conservation efforts, threatening endangered species, biodiversity and overall ecosystem health.

Outbreaks may also affect First Nations communities for whom wild bird species are of cultural significance and may also provide an important seasonal food source.

Tourism could be impacted through shifts in public attitudes but also via the disease itself and the response (such as restricted access to certain areas or sites). Recreational duck-hunting and associated activities may also be affected where the availability of, or access to, hunted species is reduced. Potentially negative cultural, community or social impacts may result from mass mortalities or when iconic species are affected.

Concerns about the zoonotic potential of AI viruses may lead to avoidance of, or restricted access to wild bird or poultry production areas. During an AI outbreak, appropriate monitoring of the virus—including its potential for sustained human-to-human transmission—will be required, along with a coordinated public information campaign.

3.3 Critical factors for an Australian response

- AI is a highly contagious infection of poultry and other birds. Clinical manifestations vary with the subtype and strain of the virus, the avian species and the age of the infected bird.
- All avian species appear to be susceptible to infection with AI viruses.
- Overseas, some subtypes of HPAI viruses have been detected in non-clinical wild birds, especially waterfowl (Giacinti et al 2024, Wight et al 2024). Recent HPAI subtypes overseas have caused mass mortalities in a wide range of both domestic and wild bird and mammalian species.
- To date in Australia, outbreaks of H7 HPAI virus have been primarily limited to poultry via mutations from LPAI virus with spread between properties largely facilitated by biosecurity deficits (e.g. through the movement of live birds, bird products (such as eggs), fomites, people, vehicles and equipment).
- Recent HPAI outbreaks in poultry overseas due to H5Nx clade 2.3.4.4b have involved direct and repeated spread from wild birds, driving both episodic resurgence and multinational transmission (Nagy et al 2023, Bellini et al 2022, Xie et al 2023).
- Previous Australian outbreaks in poultry have been resolved using response strategies founded on biosecurity, early detection, rapid containment, and depopulation. In Eurasia, these strategies do not seem to be as effective against clade 2.3.4.4b, where the emergence of a reservoir in wildlife suggests a possible enzootic status (Pohlmann et al 2022, Ramey et al 2022).
- Development in Australia of a similar situation (i.e. ongoing bidirectional transfer between wild birds and poultry) may require consideration of additional preventive and control strategies.
- Environmental factors, such as time of year and weather patterns (e.g. high rainfall, drought), appear to be important. AI viruses are readily inactivated by heating and remain viable for longer in cold and humid environments.
- Changing public perceptions of mass animal destruction, together with the challenges of rapid animal destruction and disposal may influence consideration of emergency or preventative vaccination.

- Zoo birds, rare bird collections, or other captive birds, may be susceptible to infection from both wild birds and commercial poultry operations (if any are in the vicinity) or from feeding of poultry and poultry products.
- Because of an increased likelihood of interaction between free-range commercial birds and wild birds or their faeces, there is an increased likelihood of AI in free-range poultry enterprises compared with housed commercial birds.
- Most commercial poultry farms in Australia have, and are currently mandated by catchment authorities to have, retention dams on the property, which encourage wild waterfowl resulting in an increased likelihood of introducing AI viruses onto such premises.
- Australian endemic LPAI viruses appear to be of distinct lineage divergent from Eurasian and North American AI viruses (Hansbro et al 2010). Some native Australian waterbird species may have an elevated risk of suffering severe disease from exotic HPAI due to genetics of their immune system (Karawita et al 2023).
- An outbreak of HPAI subtype H5N1 clade 2.3.4.4b may make compartmentalisation or zoning challenging, particularly where wild bird infection is confirmed in Australia. The need for, and maintenance of, heightened biosecurity and surveillance of sufficient intensity and duration to prevent introduction of disease into, or spread within zones or compartments, may be challenging to maintain and demonstrate to trading partners.

4 Policy and rationale

4.1 Introduction

The reporting requirements and response to detection of influenza A viruses in birds varies based on the virus pathogenicity and subtypes, but also on the species and category of birds in which the virus is detected. This manual outlines four key categories of birds (see Glossary): poultry (commercial), poultry (non-commercial), captive birds, and wild birds. These, along with the virus pathogenicity and subtype, are referred to throughout the manual in describing the response to an outbreak of influenza A in birds in Australia.

4.1.1 Australian and international reporting requirements

Any suspicion or detection of infection with influenza A viruses in birds is notifiable under Australian reporting requirements, as it is listed as a nationally notifiable disease.

Infection with HPAI viruses is a WOAHA-listed disease. The specific WOAHA notification requirements are outlined below (Section 4.1.1.1). Not all influenza A viruses, which would be notifiable in Australia, are notifiable to WOAHA.

The Australian response policy for AI also includes H5/H7 LPAI, for which WOAHA notification is not typically a requirement.

Where there is an active response to a detection of AI in Australia, surveillance will be undertaken for:

- resolving infected premises and declared areas which may include both HPAI and H5/H7 LPAI
- proof of freedom (H5/H7 LPAI or HPAI).

Any detection of AI occurring from these activities would be notified through the EAD response arrangements. The information required for provision to WOAHA will be determined by the Australian Chief Veterinary Officer (CVO).

4.1.1.1 WOAHA notifications

In accordance with the WOAHA *Terrestrial animal health code* Article 10.4.1 (accessed February 2024)⁵⁸:

- HPAI means an infection of poultry* by any influenza A virus that has been determined as high pathogenicity in accordance with the *WOAHA Manual of diagnostic tests and vaccines for terrestrial animals*.
- An occurrence of infection with a HPAI virus is defined by the isolation and identification of the virus or the detection of specific viral ribonucleic acid, in one or more samples from poultry*.

Infection with other influenza A viruses of avian host origin is notifiable to WOAHA as follows:

- A sudden and unexpected increase in virulence of low pathogenicity avian influenza (LPAI) viruses in poultry* is notifiable as an emerging disease in accordance with Article 1.1.4
- Infection of domestic and captive wild birds with LPAI viruses having proven natural transmission to humans associated with severe consequences, are notifiable in accordance with Article 1.1.6

⁵⁸ www.woaha.org/en/what-we-do/standards/codes-and-manuals/terrestrial-code-online-access/?id=169&L=1&htmlfile=chapitre_avian_influenza_viruses.htm

- Infection of birds other than poultry*, including wild birds, with influenza A viruses of high pathogenicity, are notifiable as a listed disease in accordance with Article 1.1.6.

A notification of infection of birds other than poultry*, including wild birds, with influenza A viruses of high pathogenicity, or of infection of domestic or captive wild birds with LPAI viruses does not affect the HPAI status of the country or zone. Countries that are members of WOAHA should not impose bans on the international trade of poultry* commodities in response to such notifications, or to other information on the presence of any non-notifiable influenza A virus in birds.

* The definition of 'poultry' adopted by the WOAHA *Terrestrial animal health code*⁵⁹ aligns with the definition of 'poultry (commercial)' (see Glossary) as used within this manual. The WOAHA definition describes poultry as: *"all birds reared or kept in captivity for the production of any commercial animal products or for breeding for this purpose, fighting cocks used for any purpose, and all birds used for restocking supplies of game or for breeding for this purpose, until they are released from captivity."*

4.1.1.2 Jurisdictional notifications

For domestic purposes, infection with all influenza A viruses in birds is nationally notifiable. The CVO of the state or territory in which the finding is made must be notified, and they in turn must notify the Australian CVO. Industry leaders would then be advised of the detection and any planned response activities.

The policy to be implemented for disease control will be informed by initial work on virus subtyping and pathogenicity completed under the Laboratories for Emergency Animal Disease Diagnosis and Response (LEADDR) program or through the CSIRO Australian Centre for Disease Preparedness (CSIRO-ACDP).

⁵⁹ www.woah.org/fileadmin/Home/eng/Health_standards/tahc/2018/en_glossaire.htm

4.1.2 Summary of policy

An outbreak of either HPAI or LPAI is defined as an infection with influenza A virus (with or without clinical signs) accompanied by confirmed laboratory diagnosis that meets the pathotyping criteria for the virus (either HPAI or LPAI).

4.1.2.1 HPAI and LPAI (H5/H7)

The default policy for an outbreak of HPAI (any subtype, including H5Nx) and LPAI (H5/H7) in commercial poultry is to contain and eradicate the disease while minimising public health, animal welfare, environmental and economic impacts. This will occur using stamping out supported by a range of other response strategies.

The response to an outbreak of HPAI (any subtype, including H5Nx) and LPAI (H5/H7) in other categories of birds (i.e. poultry (non-commercial) and captive birds) and any strategies used will depend on the assessed risk (which will include consideration of the overall response objective, the risk of disease spread to humans and other susceptible species, and animal welfare).

A combination of strategies may be employed in a response, including those outlined below:

- an *immediate epidemiological assessment* of the situation
- *stamping out* by destruction, disposal and decontamination of birds and contaminated avian products on infected premises (IP) to remove the source of infection. Pre-emptive destruction may occur on dangerous contact premises (DCPs) based on risk assessment
- *biosecurity controls such as quarantine and the use of declared areas and movement controls over properties* — this includes quarantine of IP, DCP, suspect premises (SP) and trace premises (TP); declaration of restricted and control areas; and restriction on movements of birds, avian products and associated items in declared areas to prevent the spread of infection
- *enhanced biosecurity* at places where birds are held
- *tracing and surveillance* to determine the source and extent of infection, and for the purposes of proof-of-freedom
- *flock or area depopulation* by pre-emptive slaughter, process slaughter or controlled marketing of birds not infected with AI, depending on information derived from tracing, surveillance and the epidemiology of the outbreak
- *a public awareness campaign* to communicate risk and promote cooperation from industry, zoos, poultry and captive bird owners and the community
- *protection of work health and safety, and public health*, in consultation with human health authorities — this will include a requirement that any personnel engaged in eradication activities take appropriate personal protective measures. Human seasonal influenza virus vaccination may also be considered to help prevent recombination of AI virus with seasonal human influenza viruses
- *vaccination* of birds.

4.1.2.2 LPAI (not H5/H7)

AI that is caused by a strain of virus that is not HPAI or LPAI (H5/H7), that is producing no or mild clinical disease in infected birds and is not genetically related to strains associated with severe clinical disease in humans in other countries, is not considered an EAD outbreak.

When the CVO of the affected state/territory determines that an infection is caused by such a virus, an epidemiological risk assessment will be carried out. It will consider the virus subtype, the species of bird/s

involved, the clinical status of the birds, and their proximity to commercial or other significant bird establishments and populations and to public amenity areas. Appropriate response measures will be implemented where a risk assessment indicates an unacceptable threat to animal or public health.

4.1.2.3 Specific considerations for HPAI H5Nx 2.3.4.4b

International experiences with H5Nx HPAI clade 2.3.4.4b are indicating that conventional control measures, such as biosecurity, stamping out and movement restrictions, while important, may be insufficient and unsustainable given the persistent threat of new incursions and the high viral load present in the environment due to the ubiquitous sources of the virus.⁶⁰

Due to the complexities and potential involvement of a wide range of species, including wildlife species, control measures may need to be adapted to minimise disease spread. A wider range of strategies than those historically used in Australia for HPAI outbreaks may need to be considered earlier in the response. These may include flock or area-wide depopulation of poultry, and emergency vaccination of poultry, captive birds, and wildlife species.

4.1.2.4 Avian influenza outbreak in wildlife

See also Appendix 4.

HPAI

In response to a finding of HPAI virus infection in wildlife, an epidemiological risk assessment of the situation will be conducted. The assessment should consider key epidemiological characteristics, such as the virus subtype, the possible source of infection, the species affected, the number, distribution and clinical status of affected animals, and their proximity to commercial poultry, public amenity areas and other at-risk wildlife populations.

Appropriate response measures will be implemented where a risk assessment indicates an unacceptable threat to animal or public health.

LPAI

If only LPAI virus infection is detected in wild birds, no further action is required. However, consideration may be given to increasing surveillance and biosecurity in poultry, captive birds, and wild birds in the immediate vicinity of the wild bird LPAI virus detection to monitor disease spread.

⁶⁰ www.woah.org/en/document/resolution-28-strategic-challenges-in-the-global-control-of-high-pathogenicity-avian-influenza/

4.1.3 Case definition

For the purpose of this manual, a case of AI is defined as laboratory-confirmed infection with AI virus in a susceptible animal with or without clinical signs.

Notes:

- Positive serology in the absence of detection of AI virus, with no clinical or epidemiological evidence supporting infection, does not constitute a definition of a case.
- AUSVETPLAN case definitions guide when a response to an EAD incident should be undertaken. AUSVETPLAN case definitions do not determine when international reporting of an EAD incident is required.
- At the time of an outbreak, revised or subsequent case definitions may be developed with the agreement of the Consultative Committee on Emergency Animal Diseases (CCEAD).

4.1.4 Cost-sharing arrangement

In Australia, HPAI caused by virus of subtypes H5 or H7 is included as a Category 2 EAD in the Government and Livestock Industry Cost Sharing Deed in Respect of Emergency Animal Disease Responses (EAD Response Agreement – EADRA). HPAI caused by a virus of subtype not H5 or H7 and LPAI caused by a virus of subtype H5 or H7 (LPAI (H5/H7)) are included in the EADRA as Category 3 diseases.

4.1.5 Criteria for proof of freedom

The WOA *Terrestrial animal health code* includes recommended criteria for demonstrating freedom from HPAI in poultry as per definitions in the code. These include the following:

- Infection with HPAI viruses is notifiable in the whole country, and all notified suspect occurrences of AI are subjected to field and, if applicable, laboratory investigations.
- An ongoing awareness program is in place related to avian influenza viruses risks and the specific biosecurity and management measures to address them, and to encourage reporting of suspicions of infection with HPAI.
- Appropriate surveillance is in place.
- All epidemiological factors are considered.

Freedom from infection with HPAI virus, for trade purposes, can be regained according to Article 10.4.6 of the WOA *Terrestrial animal health code*.

There are no WOA recommendations to provide evidence of freedom from LPAI, nor from HPAI in birds other than 'poultry', as referred to in this manual as poultry (commercial).

Evidence of absence of HPAI or H5/H7 LPAI in poultry or captive birds in a declared area can be established by passive surveillance and active surveillance (using both targeted and random sampling) to determine the time that has elapsed since the area's last reported case, and the resolution of high-risk premises (i.e. IPs, DCPs, dangerous contact processing facilities (DCPFs), SPs, TPs and approved disposal sites (ADSs)). Further evidence of freedom is provided by continued passive surveillance (investigation of all suspect clinical cases, with negative results) in both previously infected and uninfected areas.

Importing countries may be prepared to accept variations from the WOH criteria and allow imports of Australian live birds, hatching eggs and avian products, as part of normal bilateral agreements, on a case-by-case basis.

Section 7 provides details on the procedures for surveillance and proof of freedom.

4.1.6 Governance

Governance arrangements for the response to EADs are outlined in the **AUSVETPLAN Overview**.

Information on the responsibilities of a state coordination centre and local control centre is available in the **AUSVETPLAN Management manual: Control centres management (Part 1 and Part 2)**.

For responses to H5 HPAI in wildlife where eradication or containment is not considered feasible, the *National Management Agreement – H5 HPAI in Wildlife (H5 NMA)* applies. The H5 NMA is outside the scope of the *AUSVETPLAN Response strategy: Avian influenza*, although some activities described in this strategy may be used to inform the response.⁶¹

⁶¹ www.agriculture.gov.au/biosecurity-trade/policy/emergency/h5-hpai-nma

4.2 Public health implications

All AI viruses have the potential to infect people. All AI infections in birds in Australia should be reported to the relevant state or territory health agency to enable assessment and advice regarding the mitigation of human health risks.

Appropriate work health and safety (WHS) measures, in line with the known or theoretical risk, should be implemented, with advice from human health authorities.

Personnel must be protected from infection with AI viruses wherever they have contact with infected animals, products, premises or environments.⁶² Personnel involved in response activities will require appropriate training and supervision to ensure that all activities are managed appropriately regarding WHS. Vaccination with the currently available seasonal influenza virus vaccine is recommended for personnel who may come into contact with the virus while undertaking response activities. This is to prevent recombination of AI virus with seasonal human influenza viruses. Personnel should also wear appropriate personal protective equipment (PPE), in accordance with national human health guidelines.

Personnel working in a response to AI may be asked to take part in monitoring by state or territory public health departments. Those who do not agree to such measures should not be engaged in activities in which they could come into contact with infected birds or materials.

⁶² www.who.int/

4.3 Control and eradication policy

As described above (see Section 4.1.2.), the default policy for an outbreak of HPAI (any subtype, including H5Nx) and LPAI (H5/H7) in commercial poultry is to contain and eradicate the disease while minimising public health, animal welfare, environmental and economic impacts. This will occur using stamping out supported by a range of other response strategies.

The response to an outbreak of HPAI (any subtype, including H5Nx) and LPAI (H5/H7) in other categories of birds (i.e. non-commercial poultry and captive birds) and any strategies used will depend on the assessed risk (which will include consideration of the overall response objective, the risk of disease spread to humans and other susceptible species, and animal welfare).

The rationale for the policy is described in more detail in the remainder of this section.

4.3.1 Epidemiological assessment

Epidemiological investigation or assessment draws on multiple sources of information to build understanding of the disease and how it is behaving in an outbreak. This helps inform response decision making.

The key objectives for an epidemiological assessment will be to identify:

- the spatial distribution of infected and non-infected bird and other susceptible animal populations (including wildlife)
- the source of infection
- the prevalence of infection
- pathways of spread and the likely size of the outbreak
- risk factors for the presence of infection and susceptibility to disease (including weather and insect populations).

Epidemiological assessment, and tracing and surveillance activities (see Section 4.3.3) in an EAD response are interrelated activities. Early findings from tracing and surveillance will be inputs into the initial epidemiological assessment (e.g. considering spatial distribution of infection). The outcomes of the initial epidemiological assessment will then guide decisions on subsequent tracing and surveillance priorities.

The outcomes of the epidemiological assessment, including molecular analysis of the virus, will also be used initially to determine the feasibility of eradication versus long-term control and to guide the selection of other appropriate response measures (including the application of movement controls) and assess the progress of disease control measures.

Ongoing epidemiological assessment that will consider the outcomes of tracing and surveillance activities is important for any EAD response to aid evaluation of the continued effectiveness and value of response measures; assess the progress of disease control measures; and contribute evidence to support any later claims of disease freedom.

4.3.2 Biosecurity and movement controls

The default policy is to implement declared areas and associated movement controls; however, variations may be considered on a case-by-case basis subject to risk assessment.

See Section 6 for recommended movement controls.

Guidance on declared areas and premises classifications can be found in the **AUSVETPLAN Guidance document: *Declared areas and allocation of premises definitions in an EAD response***.

4.3.2.1 Biosecurity controls (including quarantine)

Biosecurity controls (e.g. quarantine) will be imposed on IPs, DCPs, DCPFs, TPs and SPs. These premises will remain under strict biosecurity control (e.g. quarantine) until their status has been further classified through risk assessment.

Biosecurity controls will be applied at the individual premises level to limit spread of the virus within the premises and between premises. Controls to prevent contact between susceptible wildlife and domestic animals should be implemented, where practicable, to avoid infection of domestic animals from wildlife and vice versa.

At the property level any factors that could attract wild birds to the premises should be addressed. Wild birds should be prevented from accessing or contaminating feed, water, litter and roosting trees on the property. Wildlife deterrents that are consistent with animal welfare guidelines could also be considered. Further detail on wild bird management is provided in Section 4.3.13.

Biosecurity controls should also be implemented at the poultry housing (shed) level. Sheds should be entered only when necessary. The clothing and footwear worn by workers entering sheds should be specific to each shed. All sheds should have a footbath that is used on entry and exit of the shed. Any means by which wild birds may enter sheds (e.g. air vents, broken roofs) should be addressed.

Further biosecurity controls, such as legislated housing orders for poultry with outdoor access, may be implemented in premises with susceptible animals within declared areas and possibly within the outside area. It should be noted that husbandry changes, including the implementation of housing orders, may lead to behavioural issues such as severe vent pecking and cannibalism within flocks. Such behavioural issues are difficult to eradicate once they become established within a flock, and mitigation measures should be implemented to reduce negative animal welfare effects.

Details on enhancing farm biosecurity practices and practising good biosecurity are available at the Farm Biosecurity website,⁶³ and in the following documents:

- *National farm biosecurity manual: poultry production*
- *National water biosecurity manual: poultry production*
- *National farm biosecurity manual for chicken growers*
- *National farm biosecurity technical manual for egg production*
- *National farm biosecurity technical manual for the duck industry*
- *National farm biosecurity manual for the ratite industry.*

⁶³ www.farmbiosecurity.com.au

The *National Wildlife Biosecurity Guidelines*⁶⁴ provide biosecurity guidance for where wild birds and other wildlife come into wildlife care.

4.3.2.1.1 Biosecurity for personnel

Care should be taken to prevent the further spread of AI virus through movements of people.

Personnel involved in handling poultry, captive birds and/or potentially contaminated items (e.g. those involved in sampling animals, products or byproducts; or in destruction, disposal and decontamination activities) on high-risk premises (IPs, DCP, DCPFs, SPs, ADSs and TPs) should be considered contaminated until they have completed appropriate decontamination procedures.

Jurisdictions should apply appropriate decontamination on exit of such premises, PPE requirements and/or stand-down times.

Recommended controls on the movements of people are provided in Section 6.4.8.

4.3.2.2 Movement controls

As a general principle, the aim of movement controls is to reduce the spread of disease between premises by preventing the movement of infected animals, infected animal products and infected vectors (where relevant for the disease), and by allowing or permitting movements that pose a minimal risk.

The use of declared areas under jurisdictional legislation provides the legal framework for disease control measures in EAD responses. All premises within the declared areas are subject to classification for disease control management and monitoring purposes. Classification of premises provides a framework for authorities to exercise legal powers over such premises, including movement controls.

Section 6.4 provides details on movement controls for live birds, eggs and egg products, bird products and byproducts, waste products and effluent, and other items that might be contaminated.

⁶⁴ www.wildlifehealthaustralia.com.au/Portals/0/Documents/ProgramProjects/National_Wildlife_Biosecurity_Guidelines.PDF

4.3.3 Tracing and surveillance

Tracing and surveillance will be conducted to determine the source and extent of infection, establish proof of freedom from the disease, and/or define zones and compartments for trade purposes.

4.3.3.1 Tracing

The first reported case (index case) may not be the primary case for the outbreak. Tracing may assist in identifying earlier cases (and the estimated date of introduction), determining the source of infection (and the estimated onset of infectiousness) and determining the extent of spread. During a normal poultry production cycle, many routine movements occur to and from poultry premises. Tracing of these movements should be prioritised according to the highest-risk activities and the known epidemiological risk factors.

Tracing periods should consider the WOAHP-nominated incubation period of 14 days, the age of the flock at risk and the duration of the outbreak to date. They may need to be varied during the response according to the measures being employed.

Tracing will be used to determine the movements of commodities and people associated with IPs, DCPs, SPs and TPs, including:

- live poultry and other captive birds
- poultry products – such as meat, eggs (fertile, table), semen, feathers and offal
- waste – such as dead birds, manure and used litter
- equipment – such as live bird transport vehicles, feed delivery vehicles, farm vehicles and equipment, contractor vehicles, catching equipment, dead bird pick-up equipment, veterinary equipment, workers' clothing, scales, loaders, crates, egg trays and fillers, pallets, trolleys, cages, and vaccination and debeaking equipment
- feed and fresh litter
- people – such as farm workers, employees, share farmers, poultry service workers, veterinary practitioners, tradespeople (e.g. electricians, plumbers), company personnel, sales representatives, vaccinators, pick-up crews, technicians, drivers (livestock, feed, egg, litter), contractors, livestock agents, relatives and other visitors.

Depending on the epidemiology of the virus involved, consideration may also be given to tracing in non-avian susceptible species (e.g. mammals).

The original source of likely introduction of the virus to the birds should be traced, as it could remain a source of further virus dissemination.

4.3.3.2 Surveillance

Surveillance will be necessary to identify the source and foci of infection, determine the extent of spread and assess the impact of control activities. The application of molecular epidemiology using sequencing technologies and phylogenetic analyses may add value to this understanding. Surveillance will also be used to define declared areas and provide evidence for proof of freedom when the outbreak is contained. It will also be required if zones are to be established for trade, or if vaccination use is implemented.

Active surveillance should be initiated as soon as HPAI or LPAI (H5/H7) is suspected. Initially this should involve identifying and mapping the location, species and flock size of commercial poultry, as well as non-commercial poultry, captive birds and wild birds if possible. Diagnostic testing for AI (at an approved

laboratory) should be considered if birds are found sick or dead, with the index of suspicion increasing if clinical signs consistent with AI are present or if seabirds, waterbirds, shorebirds or birds of prey are affected.

Intensive active surveillance aims to identify potential new cases of AI. Because of the risk of spread of virus by personnel, equipment and vehicles, the following measures could be adopted to enable continuing surveillance and reduce the need for multiple farm visits by inspectors and other authorised personnel to premises in the restricted area (RA) and control area (CA):

- monitoring of dead birds (poultry, captive and wild birds) – pick-up and transport to a laboratory for suspicious cases
- reporting on commercial poultry flock health and production
- telephone surveys.

Field surveillance visits to any potential new cases requiring investigation should then be arranged.

Sentinel animals may be used on decontaminated IPs as part of demonstrating effective cleaning and disinfection before full restocking of the premises, but are not a WOAHP requirement (see also Section 7.1). Additional guidance, including the frequency of sampling and numbers of birds to test under cage, barn and free-range situations, are detailed in Section 7.1.1.2.

Passive surveillance will also be conducted to complement the active surveillance, and may include poultry, captive and wild birds. This involves investigation of disease reports (i.e. SP) from the public, farmers, veterinarians, zoos, captive bird owners or wildlife managers. It includes the following:

- All reports of a decline in the health of birds (e.g. increase in mortality or morbidity consistent with clinical signs of AI; significant decline in feed or water consumption, or egg production) should be investigated.
- Samples from any captive or wild birds that die in the RA (or CA/other areas to support surveillance strategies) should be checked for suspicious gross AI lesions, and specimens should be submitted to approved laboratories for diagnostic testing.

Active and/or passive monitoring and surveillance of disease consistent with influenza in other susceptible species (e.g. both domesticated and wild) may be considered, using a risk-based approach to determine possible transmission mechanisms, incidental hosts and virus reservoirs that may warrant management or control.

Surveillance of wild birds to determine their potential involvement in dissemination of the disease may also be implemented. Surveillance in wild birds may be passive, based on reports of clinical disease and/or mortalities, or active, particularly where there are opportunities to work in with existing surveillance activities such as the National Avian Influenza Wild Bird Surveillance Program⁶⁵, duck hunting, and/or research sampling of live birds.

See Section 2.2 for more information on susceptible species.

Monitoring of human health, especially in people involved in on-property response operations, may be necessary. It should be undertaken with departmental health authorities.

⁶⁵ <https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/Wild-Bird-Surveillance>

4.3.3.2.1 Restricted area

As a priority, clinical investigation, and appropriate sampling and testing should be undertaken on all DCPs and SPs. Where findings from clinical examination and diagnostic testing are negative, ongoing surveillance (such as telephone and/or field surveillance visits) should be conducted on a regular basis (e.g. every other day) to confirm absence of disease. Testing of routine mortalities (dead bird pick-up or swabbing) may be a valuable component of monitoring.

Active surveillance may be conducted on the at-risk poultry and captive bird populations based on a risk assessment. This may include health monitoring by:

- twice weekly (or more frequent, if needed) reporting by relevant personnel (e.g. poultry and captive bird owners and handlers, veterinarians, zoo personnel, waste removalists and dead bird pick-up personnel)
- weekly tracheal or oropharyngeal and cloacal swabbing of dead birds for testing by PCR of the commercial poultry flocks in the RA, based on a risk assessment of the epidemiological picture.

4.3.3.2.2 Control area

Flock health could be monitored by:

- follow-up of any unusual disease conditions, including in wild birds
- weekly laboratory testing (PCR and other tests, as appropriate) of meat poultry and commercial spent hens and ducks at abattoirs
- weekly telephone surveillance of owners, managers or veterinarians of susceptible flocks, including those of captive birds
- weekly reporting on flock health and production parameters for commercial flocks
- appropriate flock health testing and reporting of sick or dead birds.

4.3.4 Zoning and compartmentalisation for international trade

Where it is not possible to establish and maintain disease freedom for the entire country, establishing and maintaining disease-free subpopulations, through zoning and/or compartmentalisation,⁶⁶ may be considered.

In the case of a limited disease outbreak, a containment zone⁶⁷ may be established around the areas where the outbreak is occurring, with the purpose of maintaining the disease-free status of the rest of the country outside the containment zone.

All zoning applications would need to be prepared by the Australian Government in conjunction with the relevant jurisdiction(s) and agreed to by the CCEAD. Zoning is usually negotiated after a disease outbreak has begun.

⁶⁶ With zoning, disease-free subpopulations are defined primarily on a geographical basis. With compartmentalisation, disease-free subpopulations are defined primarily by management practices (such as the biosecurity plan and surveillance practices of enterprises or groups of enterprises).

⁶⁷ The WOA defines a 'containment zone' as an infected zone within a previously free country or zone, which includes all suspected or confirmed cases that are epidemiologically linked and where movement control, biosecurity and sanitary measures are applied to prevent the spread of, and to eradicate, the infection or infestation.

Compartmentalisation applications typically need to be negotiated before an outbreak occurs, and will require input from the relevant industries.

Recognition of both zones and compartments must be negotiated between the Australian Government and individual overseas trading partners. Zoning and compartmentalisation would require considerable resources that could otherwise be used to control an outbreak. Careful consideration will need to be given to prioritising these activities, because the resulting competition for resources could delay the quick eradication of the disease and recognition of disease freedom.

Agreements between trading partners take time to develop, consider and finalise, because of the need to provide detailed information on activities such as biosecurity, surveillance, traceability and diagnostics to support the approach that is developed. An importing country will need assurance that its animal health status is not compromised if it imports from an established disease-free zone in Australia. Trading partners may not accept a zoning or compartmentalisation proposal, regardless of the information provided. Eradication of disease may be achieved before zoning or compartmentalisation applications are finalised.

The WOAH guidelines for zoning and compartmentalisation are in Chapter 4.4 and Chapter 10.4 of the *WOAH Terrestrial animal health code*.

4.3.5 Vaccination

Vaccination has not been necessary, nor available, in Australian outbreaks to date, but its usefulness has been demonstrated in some overseas outbreaks as part of comprehensive control measures (Capua et al 2003, EFSA 2023, Hautefeuille 2020, Islam et al 2023, Liu et al 2020, Swayne et al 2011, WOAAH 2023).

As of March 2025, one avian influenza (H5N1) vaccine is registered for use in Australia for which the Department of Agriculture, Fisheries and Forestry maintains an import permit. Three active constituents (H7N1, H5N9, H5N2) have been approved by the Australian Pesticides and Veterinary Medicines Authority (APVMA). If any vaccine or active constituent were to be considered for use in Australia, the APVMA would have to be consulted to determine the conditions and implications of use, as well as any other regulatory requirements.

The UN FAO and WOAAH '*Global Strategy for the Prevention and Control of High Pathogenicity Avian Influenza (2024–2033)*' details the recommended approach for the management of HPAI, including the use of vaccines *en masse*. This departs from the previous global strategy.

The use of a vaccine in Australia will be under the control of CVOs in each state or territory. If vaccination is considered for control purposes by the affected state or territory CVO, it will be supported by intensive liaison across all poultry and captive bird sectors, and by public awareness programs.

See also the **AUSVETPLAN Guidance document: *Risk-based assessment of disease control options for rare and valuable animals*** Section 4.2 *Assessment specific to request for protective vaccination* and the national operating policy and procedures for the use of avian influenza (AI) vaccine in the event of an AI outbreak in Australia⁶⁸. The Global Strategy for the Prevention and Control of High Pathogenicity Avian Influenza (2024–2033) also describes the use of vaccination where other methods alone are not achieving sufficient control.⁶⁹

See also Section 2.7.

4.3.6 Treatment of infected animals

Treatment of infected birds in Australia is generally not permitted; however, supportive treatment may be considered on a case-by-case basis by the jurisdiction in instances when stamping out is not employed.

4.3.7 Treatment of animal products and byproducts

All products from infected birds, and other items that contained, transported or came into contact with such products or byproducts should be considered to be contaminated with AI virus.

Eggs produced by the infected flock during the tracing period need to be destroyed and disposed of. Egg pulping is not a common method used for treating eggs during HPAI outbreaks; however, the decision of egg pulping as an effective treatment could be considered on a case-by-case base following a risk assessment indicating an acceptable (low) level of risk.

⁶⁸ www.agriculture.gov.au/sites/default/files/documents/ahc-avian-influenza-vaccination-policy-birds.pdf

⁶⁹ Global Strategy for the Prevention and Control of High Pathogenicity Avian Influenza (2024–2033) – Achieving sustainable, resilient poultry production systems

4.3.8 Destruction of animals

4.3.8.1 HPAI and LPAI (H5/H7)

Stamping out is the default control measure for HPAI and LPAI (H5/H7) on infected premises with commercial poultry, including the destruction of all susceptible poultry species on the premises. Destruction of non-commercial poultry and captive birds on such premises will be subject to risk assessment.

Stamping out is considered on a case-by-case basis for HPAI and LPAI (H5/H7) on infected premises where commercial poultry are not present (i.e. those with only non-commercial poultry and/or captive birds). It will be considered where the risk of spread to other premises cannot be adequately mitigated via other biosecurity/response measures, and/or if public health and animal welfare risks cannot be mitigated.

Destruction of wild birds on any premises would only be considered for welfare purposes.

Pre-emptive destruction of susceptible species on dangerous contact premises will be considered and assessed on a case-by-case basis following risk assessment. Considerations in such an assessment will include the measures used to control the incident, the success of other activities and the epidemiology of the incident.

Any further exemptions to stamping out will be assessed on a case-by-case basis following risk assessment.

Where destruction is to be undertaken it should be completed as rapidly as possible to:

- alleviate suffering of infected animals
- reduce shedding of virus and spread of disease
- minimise the potential for mutation
- decrease the likelihood of emergence of new viral subtypes that may sustain animal-to-animal or person-to-person transmission.

The speed in which response measures, including stamping out and movement controls, occur is very important for the rapid control of an outbreak.

Destruction plans should be developed for each premises on which animals are to be destroyed. Destruction is subject to approval by the jurisdictional CVO and preferably occurs without moving birds from the premises. The animal welfare aspects of the different destruction options should be considered when selecting a method. Destruction methods are described in the **AUSVETPLAN Operational manual: *Destruction of animals***.

Zoonotic potential and human safety operational risks must be considered with methods that require manual handling of infected birds. Handling dead birds produces less airborne contamination than catching and handling live birds, thereby reducing the exposure of workers to virus and making working in the recommended protective equipment more tolerable. However, handling dead birds in cages while rigor mortis is present may create significant challenges for animal removal.

Airborne dispersal of virus into the environment can be minimised during destruction by ventilation adjustments and covers on shed openings. Depopulation activities should occur inside sheds as far as practicable. Movement of infected or potentially infected birds for destruction, if required, should remain within the immediate vicinity of the shed.

Consideration should be given to prioritising destruction of infected birds and birds where welfare is compromised (e.g. due to overcrowding, lack of available food or water) over non-clinically affected in-contact/at risk birds where possible and as relevant.

4.3.8.2 LPAI (non H5/H7)

Stamping out may also be considered for LPAI (not H5/ H7) on a case-by-case basis, where the strain or a closely related one is associated with disease in humans in Australia or globally, or where molecular markers indicate an increased likelihood of transmission to and between mammals.

4.3.9 Process slaughter and controlled marketing

Process slaughter may be applied to premises of relevance (PORs) or at-risk premises (ARPs) and other premises as determined by a risk assessment. This might be appropriate where large numbers of poultry must be destroyed quickly, and the product is deemed fit for human consumption or is treated to inactivate any AI viruses as a precautionary measure. Process slaughter may limit the economic impact of the outbreak and minimise disruption to food supply.

All process slaughter activities should be subject to risk assessment and may include consideration of flock laboratory diagnostic testing and application of appropriate biosecurity controls.

Where risk assessment determines that process slaughter poses a low level of risk to humans and other poultry associated with movement, slaughter and processing and thus is appropriate, the following controls may apply, in addition to any other requirements as deemed necessary:

- no clinical signs associated with AI in birds at time of transport
- all birds on site to be processed
- all personnel handling and slaughtering birds to wear PPE, including goggles and masks
- trucks moving birds from farm to processing be covered in fine mesh to prevent escape of feathers from birds
- transport route to avoid known poultry establishments and suburban areas
- feathers from processed birds not to be used for human use (e.g. pillows) without further processing
- complete farm decontamination prior to restocking.

Birds that have recovered from AI infection are likely to test PCR positive for several weeks following active infection and thus a requirement for birds to test PCR negative prior to transport may not be required.

Controlled marketing of poultry on ARP or POR that are not infected but are at risk may also be undertaken, provided that a risk assessment indicates that the birds do not pose an unacceptable risk of disease spread, or a work health and safety risk, and that effective biosecurity controls can be imposed.

There are examples of the successful use of process slaughter within the Australian context. Examples include a commercial breeder flock in New South Wales in 2010 (H10N7, LPAI) and a small commercial multi-aged duck flock in New South Wales in 2012 (H4N6, LPAI).

In the case of the former, birds were processed only from seronegative flocks on the infected premises as reported by Arzey et al (2012). In this instance, process floor workers were subsequently infected with H10N7, developing mild respiratory signs. Of note was that due to low perceived risk (negative serology), these workers did not wear facial PPE (goggles and mask).

In the case of the latter, subsequent to risk assessment (deemed low level of risk to humans and other poultry associated with movement, slaughter and processing) process slaughter of ducks from the farm was allowed, requiring the controls as described in detail above. PCR testing with a negative result was not required for each batch prior to movement for processing based on risk assessment that deemed a low food safety risk, provided that all birds were non-clinical at the time of transport.

4.3.10 Disposal of animals and animal products and byproducts

One of the major objectives of the eradication program is prompt and effective disposal of contaminated material that cannot be effectively treated (e.g. dead birds, eggs, litter, manure, fresh or frozen carcasses, plant and equipment, and building materials).

Disposal of large numbers of birds in a short time presents environmental and logistical problems. A poultry shed full of meat birds close to market weight contains about 75–90 tonnes of organic material, of which 75% is water.

Disposal may be either on or off the IP or DCP. The best method should be determined by risk assessment, considering factors such as the proximity to appropriate disposal sites, the risk of virus spread via transport, and the impact on businesses and the community. Available methods of disposal are described in the **AUSVETPLAN Operational manual: Disposal**. The most likely on-site disposal methods include composting and deep burial. Off-site disposal by burial at a common site, incineration, composting or rendering could be used if on-site disposal is not suitable or practical.

If infected material must be transported for disposal, particular attention should be paid to preventing the spread of the virus. For example, truck body trays must be leakproof, and all loads must be carefully covered to ensure that material cannot escape.

Treatment and disposal of manure and litter will depend on the premises and circumstances and will require assessment of the most appropriate location and means of disposal. The **AUSVETPLAN Operational manual: Disposal** and **AUSVETPLAN Operational manual: Decontamination** must be consulted when deciding on the most appropriate means of disposal and decontamination respectively.

See Section 4.3.13 for information on wild bird management.

4.3.11 Decontamination

Decontamination of contaminated premises and fomites (e.g. clothing, footwear, non-disposable equipment) is a critical part of the response to avian influenza. Decontamination plans should be developed for each premises to be decontaminated.

The decision to decontaminate or destroy and dispose an item is made on a case-by-case basis. Destruction and disposal are usually chosen for items that would cost more to decontaminate than they are worth; however, for items that cannot be safely or adequately cleaned, the item may be valued and included as part of owner compensation.

The lipid in the AI virus envelope makes the virus highly susceptible to a wide range of chemical decontaminants including soaps and detergents. The Australian Pesticides and Veterinary Medicines Association (APVMA) permit PER89609 details the relevant chemicals approved for use in an AI outbreak.

Contaminated sites should be decontaminated following removal of animals and disposal of contaminated materials, with completion of the following:

- Contaminated buildings, machinery and equipment, and vehicles must be decontaminated.
- Contaminated fomites, such as clothing, footwear, crates, feed bags, egg fillers and other equipment, should be decontaminated, if possible, or disposed of in a biosecure manner.
- Manure and litter on IP and DCP must be decontaminated (e.g. through composting processes to pasteurise the material) or disposed of in a biosecure manner.
- Decontamination should include standard insect vector and rodent control measures to minimise mechanical spread of the virus to nearby premises.

People exiting an IP, DCP, DCPF or SP need to follow strict decontamination procedures if AI virus is to be contained (see the **AUSVETPLAN Operational manual: Decontamination** and the nationally agreed standard operating procedure *Personal decontamination – entry and exit procedures*⁷⁰). In addition, it needs to be ensured that products from other premises in the area of infection do not transfer AI virus before infection is diagnosed on those premises.

Guidance on decontamination, including avian influenza specific information on thermal decontamination, can be found in the **AUSVETPLAN Operational manual: Decontamination**.

4.3.12 Animal welfare

Guidance on managing livestock welfare can be found in the **AUSVETPLAN Operational manual: Livestock welfare and management**. Because morbidity resulting from AI may be high, close monitoring and careful management of animal welfare on affected premises will be required. It is critical to continue to supply adequate food, water and ventilation and veterinary care and advice as required. Rapid, humane destruction of infected flocks or individual animals may need to be prioritised according to the severity of clinical signs or of compromised animal welfare.

⁷⁰ <https://animalhealthaustralia.com.au/nationally-agreed-standard-operating-procedures/>

4.3.13 Wild bird management

To minimise the risk of infection of poultry and captive birds from wild birds (and vice versa), high-level enhanced biosecurity must be practised, in addition to general industry recommendations.^{71,72 73,74}

Wild birds cannot be completely controlled outside of poultry sheds, so sheds must be considered as a critical biosecurity control point. The design of the poultry operation needs to consider mitigation measures to deter wild birds with a focus on wild waterfowl. During poultry or captive bird eradication procedures, it is essential that quarantined and other bird houses, litter, waste, compost piles and contaminated sites are bird-proofed to prevent access by wild birds and mammals.

Steps should be taken to make IPs and DCPs less attractive to wild birds, including cleaning up feed spills. Netting or draining of dams are unlikely to be practicable but are feasible under certain circumstances and should be evaluated as options. The use of drones and laser lighting and electromagnetic field deterrent technologies have been demonstrated to discourage wild birds from landing and settling on range areas and water bodies.

Wild bird management must continue long after destruction, disposal and decontamination for poultry and captive birds have been completed, and throughout targeted or passive surveillance and epidemiological investigations, because wild birds present a source of reinfection of a site, particularly if they have been implicated in the initial index case.

Scientifically, destruction of wild birds is not supported. Experts do not recommend the lethal removal of wild birds to prevent the spread of LPAI or HPAI. Use of lethal methods is neither practical nor environmentally or ethically sound because of the high number and constant movement of wild birds.⁷⁵ Some animals may also be of high conservation value or rare and the loss of these animals through disease management would also likely be considered to have negative impacts on conservation, community or tourism. Also, bird species may have critical cultural and/or economic importance to First Nations communities.

Where there are also significant mortality events in wildlife during an outbreak of AI, the evidence to support the benefits of removing wildlife carcasses is still very limited; however, it has been shown to lower mortality in the remaining population under some circumstances, especially when removal starts as early as possible (Furness et al 2023, Knief et al 2024). It should also be considered that removal and disposal of wildlife carcasses may disturb animals (which can be of particular concern in breeding colonies), contribute to spread of infection via people, equipment and vehicles if not undertaken in a biosecure manner, present difficulties in resourcing and logistics for collection and disposal of carcasses in a timely and biosecure manner (particularly if there are large numbers), and create human health risks with handling of carcasses. Decisions around the removal of wildlife carcasses should be determined based on a risk assessment⁷⁶ that considers risk factors such as those described above.⁷⁷ If it is determined that carcass removal is an option, consideration should also be given to monitoring and evaluating the outcomes to help contribute to the evidence base for this strategy.⁷⁸

Habitat destruction or chemical disinfection of environments should not be considered as HPAI control measures in wildlife populations, as these are considered potentially counterproductive (e.g. likely to

⁷¹ www.agriculture.gov.au/sites/default/files/sitecollectiondocuments/animal-plant/pests-diseases/biosecurity/poultry-bio-manual/poultry-biosecurity-manual.pdf

⁷² www.farmbiosecurity.com.au/wp-content/uploads/2022/10/National-Farm-Biosecurity-Manual-for-Chicken-Growers_Oct2022.pdf

⁷³ www.farmbiosecurity.com.au/wp-content/uploads/2020/04/Farm-Biosecurity-Manual-for-the-Duck-Meat-Industry_2020.pdf

⁷⁴ www.farmbiosecurity.com.au/wp-content/uploads/2023/10/National-Farm-Biosecurity-Manual-for-the-Ratite-Industry.pdf

⁷⁵ www.fao.org/4/y5537e/y5537e0b.htm

⁷⁶ <https://epicscotland.org/resources/epic-veterinary-risk-assessment-wild-bird-carcass-collection-in-the-event-of-mass-mortality-due-to-suspected-highly-pathogenic-avian-influenza-hpai/>

⁷⁷ www.cms.int/sites/default/files/publication/avian_influenza_2023_aug.pdf

⁷⁸ www.agriculture.gov.au/sites/default/files/documents/national-principles-wildlife-carcass-management.pdf

disperse wildlife populations, potentially assisting in spreading the virus further), not effective for disease control and may be directly harmful to the environment.^{79,80}

Table A4.1 (Appendix 4) outlines the control options for several AI scenarios where there are detections of HPAI or LPAI in wild birds, without detections in poultry or captive birds. For further information on the overall framework for the management strategies and control procedures for wild animals during an emergency animal disease, see the **AUSVETPLAN Operational manual: *Wild animal response strategy***.

4.3.14 Vector management

Rodents, wild and domestic animals (e.g. dogs and cats), and flying insects may act as mechanical vectors for AI virus. Appropriate biosecurity measures must be implemented to ensure that these potential vectors cannot access or remove material from bird areas or sheds or composting or disposal sites.

Rodent and insect control measures, such as baiting, chemical control or vermin-proofing, should be implemented as practicable for each site.

For further information on the overall framework for the management strategies and control procedures for wild animals during an emergency animal disease, see the **AUSVETPLAN Operational manual: *Wild animal response strategy***.

4.3.15 Public awareness and media

Public awareness programs will aim to:

- maximise reporting of suspect cases by veterinarians, owners of poultry and other birds, and managers of premises containing birds
- gain community cooperation
- build confidence in disease control measures.

The *Biosecurity incident public information manual*⁸¹ provides a guide for undertaking activities associated with public information management.

Details on enhancing farm biosecurity practices and practising good biosecurity are provided in Section 4.3.2.

In addition to biosecurity messaging, a media campaign should emphasise the importance of poultry producers, bird owners, zoo personnel, wildlife managers and the public reporting suspicious clinical signs and unusual deaths promptly.

Details of any imposed movement controls need to be made available and clearly explained.

Human health considerations need to be highlighted. People should be directed to human health authorities for targeted information on hygiene practices when dealing with poultry and poultry products.

⁷⁹ www.cms.int/sites/default/files/publication/avian_influenza_2023_aug.pdf

⁸⁰ www.fao.org/animal-health/situation-updates/global-aiv-with-zoonotic-potential/recommendations/en

⁸¹ <https://animalhealthaustralia.com.au/bipim/>

Information must be provided to the public to address concerns about the safety of poultry products and effects on the supply chain.

4.3.16 Other strategies

Restocking of flocks or areas should only be undertaken after a risk assessment and consideration of the epidemiological situation. No restocking should take place before the outbreak has been brought under control in the area where infection was widespread.

Before full restocking, sentinel birds may be used to determine the effectiveness of decontamination measures. This approach may delay full restocking but has the advantage of lowering costs should a fully restocked premises become infected as a result of inadequate decontamination.

It is vital that sentinel birds have ample opportunity to be exposed to AI virus should it remain in the decontaminated area. In cage layer operations, this may require allowing access of sentinel birds to cages, the floor and manure collection areas. In free-range operations, access to all production and housing areas (e.g. laying areas, feeders, night housing) must be allowed.

When determining the time between decontamination and restocking of premises with sentinels or full restocking, virus viability outside the host (see Section 2.4.2.2 and Appendix 3) should be considered. This will take into account factors influencing virus viability, including temperature, humidity, salinity, pH, surface type, ultraviolet light and chemical application (see Appendix 3). Economic and social factors associated with delayed recommencement of business operations should also be considered.

Historically, restocking of premises with sentinel birds or full restocking has not been allowed until at least 14 days following cleaning and disinfection. The basis for this is unclear because limited information is available on virus viability on surfaces that are likely to be found in poultry sheds. Alternative time periods for restocking of IPs and DCPs may be appropriate and would be informed through risk assessment.

4.3.17 Stand-down

Stand-down of the response will occur when the National Management Group (NMG) formally declares that the outbreak is over. This may be when it decides (on advice from the CCEAD) that:

- AI has been eradicated or
- eradication is no longer considered feasible or
- following completion of the transition to management (T2M) phase.

Controls may still be in place at the jurisdictional level during the T2M. Additional information on T2M can be found in the **EADRA**.⁸²

Additional information on the stand-down of EAD responses can be found in the **AUSVETPLAN Management manual: Control centres management (Part 1)**.

If necessary, relief and recovery activity will continue after disease control and eradication operations have wound down.

⁸² <https://animalhealthaustralia.com.au/eadra/>

4.4 Other control and eradication options

4.4.1 Rare or valuable poultry and captive birds

When an IP or DCP (based on risk assessment) on which stamping out is to be applied contains rare or valuable poultry, or captive birds, the primary objective remains eradication of AI virus. An alternate approach may be considered following a risk assessment, in which uninfected birds or flocks on these properties are exempt from destruction. This would only apply if the risks were assessed as acceptable, considering factors such as biosecurity, animal welfare, public health, potential for onward transmission, likelihood of exposure, movement controls, ongoing tracing and surveillance, and timeliness in achieving disease eradication.

An exemption from stamping out (on premises where it is to be implemented) may be considered for:

- rare breeds that are listed on the Rare Breeds Trust of Australia priority list, or other lists of similar or academic recognition
- endangered or rare species – for example, species listed by the Convention on International Trade in Endangered Species, or species on the International Union for Conservation of Nature Red List of Threatened Species
- birds with specialised skills or attributes (e.g. animal actors, airport bird control raptors, public performance birds)
- irreplaceable birds (which cannot be replaced because of limited global availability or import restrictions)
 - birds with unique genetics – that is, genetically distinct birds, with a documented breeding history, that are in a viable reproducing population or part of a collective breeding situation (sufficient to maintain genetic variation). In some cases, individual birds may participate in geographically dispersed breeding programs, which may include some grandparent flocks. Sufficient proof would be required to demonstrate genetic uniqueness
 - birds in research colonies
- private captive bird collections, including pet birds
- birds in recognised zoos and wildlife sanctuaries and public aviaries
- birds in facilities breeding native wild species for release.

Subject to a risk assessment and in consultation with human health authorities, rare or valuable birds may be exempted from destruction, either temporarily or permanently, if they:

- are showing no clinical signs of AI virus infection (LPAI H5/H7) and are tested and found to be free from active infection, where practicable (testing may not be practicable for some zoo birds)
- are placed under quarantine
- are subject to surveillance; such surveillance, if part of the eradication campaign, would be subject to cost-sharing arrangements
- are covered by an approved biosecurity plan developed by the owner or veterinarian, in consultation with jurisdictional (including health) authorities
- are subject to an acknowledgment from those looking after the birds that they are aware of the risks to human health, and agree to use PPE when handling the birds or working in their enclosures, and avoid close contact with the birds
- do not pose a risk to commercial enterprises or other susceptible animal populations.

Vaccination for such birds may be considered if a case can be made for protection of high-value birds at risk of infection. The use of AI vaccines in Australia is only permitted in accordance with a decision by the CCEAD, but with control of use under the jurisdictional CVO, with the priorities being to stop spread of the outbreak and to protect rare, endangered and valuable birds (targeted vaccination). A vaccination policy for rare, protected and valuable avian species ('non-poultry') has been developed⁸³ (see also **AUSVETPLAN Guidance document: *Risk-based assessment of disease control options for rare and valuable animals*** Section 4.2 *Assessment specific to request for protective vaccination*). Identification of individual birds may be necessary.

Given the value of some zoo birds, AI vaccination may be an appropriate preventive measure, although it may not increase their protection if they are already housed in biosecure units. An application will need to be made for vaccination, declaring that the flock or bird is of exceptional value, and that certain conditions and expenses can be met. Other requirements are listed in the policy for rare, protected and valuable avian species.

In the event of a diagnosed or reported outbreak of LPAI (not H5/H7) in a rare or valuable bird flock, management of the flock should be subject to risk assessment, which will consider the risk to public health, in consultation with human health authorities.

For further information, refer to the **AUSVETPLAN Guidance document: *Risk-based assessment of disease control options for rare and valuable animals***.

4.4.2 Funding and compensation

Details of the cost-sharing arrangements can be found in the Government and Livestock Industry Cost Sharing Deed in Respect of Emergency Animal Disease Responses.⁸⁴ Details of the approach to the valuation of, and compensation for livestock and property in disease responses can be found in the **AUSVETPLAN Operational manual: *Valuation and compensation***.

⁸³ www.agriculture.gov.au/sites/default/files/documents/ahc-avian-influenza-vaccination-policy-birds.pdf

⁸⁴ <https://animalhealthaustralia.com.au/eadra/>

5 Areas and premises classifications

Information on areas and premises classifications is provided in the **AUSVETPLAN Guidance document: *Declared areas and allocation of premises definitions in an EAD response.***

The size and boundaries of the restricted area (RA) and control area (CA) should be risk-based, taking into consideration the epidemiology of the disease. Criteria for risk assessment include but are not limited to:

- known human-assisted and natural movements of birds and risk materials (e.g. tracing data)
- the location, distribution and, where known, premises/area classification of populations of susceptible animals
- biosecurity practices
- the location of key elements of the industry supply chain
- the impacts of disease control measures compared with the expected benefits of disease control.

6 Movement controls

6.1 Principles

General principles for quarantine practices and movement controls for managing emergency animal diseases (EADs) are provided in the **AUSVETPLAN Guidance document: *Movement controls***.

6.2 Guidelines for issuing permits

Recommended biosecurity and movement controls provide guidance on which movements can be allowed and under what conditions. This is based on an analysis of the disease risks that are presented by a specific movement, of a specific commodity, at a specific time during the EAD response phase. This response strategy indicates whether a proposed movement is:

- allowed (under normal jurisdictional, including interstate, requirements)
- prohibited – except under the conditions of a general, special or emergency permit
- prohibited.

Permits may not be available until the relevant CVO provides approval for movements, and this may not be available in the early stages of a response.

Guidelines for issuing permits are provided in the **AUSVETPLAN Guidance document: *Movement controls***.

Movements not reflected within this manual may be considered by the relevant jurisdictional CVO on a risk-assessed case-by-case basis.

6.3 Types of permits

Permits are either general or special. Emergency permits are a form of special permit (see also Glossary).

They are legal documents that describe the animal(s), commodities or things to be moved, the origin and destination, and the conditions to be met for the movement. Both general and special permits may be in addition to documents required for routine movements between or within jurisdictions (e.g. health certificates, waybills, consignment notes, National Vendor Declarations (NVDs)).

Details on permit types are provided in the **AUSVETPLAN Guidance document: *Movement controls***.

6.4 Recommended movement controls

Refer to the **AUSVETPLAN Enterprise manual: *Poultry industry*** for detailed industry information.

Processing facilities refer to facilities/premises where any of the following activities occur prior to retail: storing, handling, washing, candling, grading, or packing eggs on behalf of others; pasteurising egg products; processing of eggs to egg products; slaughter of live birds; and further processing of bird products subsequent to slaughter.

Movement controls are commensurate with the determined risk, taking into account the need to minimise impacts on business continuity, and return to normal business and trade as soon as possible.

6.4.1 Poultry and captive birds

Table 6.1 describes the recommended movement controls for live poultry and captive birds (including chicks, pullet hens, broilers, breeders) within and between areas.

Movement of sentinel birds to IPs following destruction, disposal and decontamination, can be considered on a case-by-case basis.

Table 6.1 Recommended movement controls for poultry and captive birds within and between areas

To→		RA						CA						OA	
From ↓		IP / DCP	SP	TP	DCPF	APF	UPF ⁸⁵	ARP	SP	TP	DCPF	APF	UPF ⁸⁵		POR
RA	IP	Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 7, 8, 10, 11)		Prohibited		Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 7, 8, 10, 11, 13)		Prohibited		Prohibited	
	DCP	Prohibited		Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11)		Prohibited		Prohibited		Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)		Prohibited		Prohibited	
	SP	Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 7, 8, 10, 11)		Prohibited		Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 7, 8, 10, 11, 13)		Prohibited		Prohibited	
	TP	Prohibited		Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11)	Prohibited	Prohibited	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited	Prohibited
	ARP	Prohibited		Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11)	Prohibited (except under GP – conditions 3, 5, 7, 8, 9, 10, 11)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12)	Prohibited	Prohibited except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)			Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 12)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12)

⁸⁵ A UPF is an abattoir, knackery, milk or egg processing plant or other such facility where the current presence of susceptible animals and/or risk products, wastes or things is unknown. UPF can be used as a default status in a response until there is sufficient information to reclassify it.

To→		RA						CA						OA		
From ↓		IP / DCP	SP	TP	DCPF	APF	UPF ⁸⁵	ARP	SP	TP	DCPF	APF	UPF ⁸⁵		POR	
CA	SP	Prohibited			Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 10, 11, 13)	Prohibited			Prohibited			Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 10, 11)	Prohibited			Prohibited
	TP	Prohibited		Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited	Prohibited	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 7, 8, 10, 11, 13)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 7, 8, 10, 11, 12)	Prohibited	
	POR	Prohibited		Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited (except under GP – conditions 3, 5, 7, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12)	Prohibited	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11)	Prohibited (except under GP – conditions 3, 5, 7, 8, 9, 10, 11)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under GP – conditions 3, 5, 6, 8, 9, 10, 11, 12)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12,)	Prohibited
OA		Prohibited		Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited (except under GP – conditions 3, 5, 6, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 7, 8, 9, 10, 11, 13)	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12)	Prohibited	Prohibited (except under SpP – conditions 3, 4, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under SpP – conditions 3, 4, 5, 7, 8, 9, 10, 11, 13)	Prohibited (except under GP – conditions 3, 5, 6, 8, 9, 10, 11, 13)	Prohibited (except under GP – conditions 3, 5, 6, 8, 9, 10, 11, 12, 14)	Prohibited (except under GP – conditions 3, 5, 6, 8, 9, 10, 11, 12)	Allowed under normal jurisdictional and interstate movement requirements	

APF = approved processing facility; ARP = at-risk premises; CA = control area; DCP = dangerous contact premises; DCPF = dangerous contact processing facility; GP = general permit; IP = infected premises; OA = outside area; POR = premises of relevance; RA = restricted area; SP = suspect premises; SpP = special permit; TP = trace premises; UPF = Unclassified processing facility

In circumstances where there is no suitable processing facility located with the RA or CA, movements to the OA for slaughter, subject to risk assessment, may be considered on a case-by-case basis.

Permit conditions for Table 6.1:

1	Direct movement to abattoir for destruction and treatment (virus inactivation) or disposal.
2	Only if on-farm destruction is not the preferred option.
3	Each load is consigned to a single premises.
4	Risk assessment – Under approval from CVO, or CVO-authorized delegate, after assessment ⁸⁶ indicates that the risk associated with the movement is acceptable within the response.
5	Travel by approved routes and no stopping en route; or travel by main roads/highways and not transiting through a property or stopping en route adjacent to a known poultry production area.
6	The dispatching and receiving premises must meet agreed biosecurity standards.
7	The receiving premises must meet agreed biosecurity standards
8	Vehicles carrying live poultry and captive birds are decontaminated after unloading and the decontamination process is verifiable. Decontamination must occur before entry to a new poultry premises.
9	Absence of clinical signs consistent with AI in all poultry on the premises of origin.
10	Any suspicious or clinical signs of AI in poultry proposed to be moved are immediately reported to the relevant jurisdiction or through the Emergency Animal Disease Hotline (1800 675 888).
11	All poultry movements must comply with state/territory legislation related to traceability requirements/standards, and be accompanied by relevant state(s)/territory(ies) movement document(s). Traceability must be maintained for a minimum of 30 days for consignments moved to another farm (or as required by jurisdictional legislation).
12	Introduced poultry are kept separate ('quarantined') for a minimum of 14 days before introduction to the flock, unless they have originated from a premises that is epidemiologically linked and with the same biosecurity status as the destination premises. Biosecurity controls are applied to personnel, equipment (fomites) and feed to eliminate contact between different biosecurity units as per the agreed biosecurity standards, together with specific biosecurity enhancements agreed by the CVO.
13	Only where there is no capacity to process in the declared area of origin.
14	Only in exceptional cases, to ameliorate animal welfare issues between epidemiologically linked premises and where the TP (origin or destination) are assessed as low risk.

⁸⁶ This may include clinical surveillance and/or diagnostic testing of poultry scheduled for movement, or background surveillance testing of 'normal', sick and dead poultry to exclude AI.

6.4.2 Carcasses

Movement of carcasses for disposal (to premises/processing facilities for treatment that will inactivate the virus, e.g. rendering) is subject to the following movement conditions.

Movements from:

- high-risk premises (IP, SP, DCP, TP) are prohibited except under special permit (subject to conditions 1, 2, 3, 4, 5 and 7)
 - DCPs and TPs are further subject to condition 6
- the RA and CA to the OA are prohibited
 - with the exception of movements from POR to the OA under general permit (subject to conditions 1, 4, 5, 6, 7 and 8)
- lower-risk premises (ARP and POR) are prohibited except under general permit (subject to conditions 4, 5, 6, 7 and 8)
- the OA into the RA or the CA are prohibited except under special permit (subject to condition 5)

Movements within the OA (unless the premises itself is subject to individual restrictions) are allowed under normal jurisdictional requirements.

Permit conditions for movement of carcasses:

1	Direct movement from premises of origin to disposal site.
2	Under approval from CVO (for emergency permits) or CVO delegate/inspector of livestock (for SpP) after assessment indicates that the risk associated with the movement is acceptable within the response. This may include laboratory testing of sick and dead poultry to exclude AI.
3	Travel by approved routes and no stopping en route.
4	Carcasses must be transported in leakproof trucks, vehicle trays or containers.
5	Vehicles and equipment carrying carcasses are decontaminated (i.e. cleaned and disinfected) after unloading and inspected/certified as such.
6	Absence of clinical signs consistent with AI in all poultry or captive birds on the premises before and on the day of dispatch. Any clinical signs in poultry or captive birds suspicious for, or consistent with, AI are immediately reported to the local control centre, state coordination centre or Emergency Animal Disease Hotline (1800 675 888).
7	Any material permitted for movement must not be brought into direct or indirect contact with susceptible livestock.
8	Multiple pick-ups are permitted only if collection points are at farm perimeter within each designated declared area.

6.4.3 Meat and meat products

Table 6.2 describes the recommended movement controls for meat and meat products within and between areas. This includes meat, whole birds and all other products recovered from the processing plant (e.g. offal, feet, tongues, oviducts, ova, frames, bones, pluck) for retail or further processing into products for human consumption or pet food.

This section does not cover movements of wild harvested meat or meat products.

Risk assessments for permit applications for movements of meat or meat products must consider:

- the likelihood that the consignment of poultry was infected at the time of processing. This will include consideration of the classification of the premises of origin of the animals, and may include testing of any animal or carcass suspected of being infected with AI to confirm or exclude AI⁸⁷
- the likelihood that meat or meat product has been cross-contaminated by infected or contaminated poultry or poultry product during processing, including aggregated product that may contain material from multiple premises. This may include testing of meat or meat products suspected of being contaminated to confirm or exclude AI
- whether product that is likely to be contaminated can be identified and traced among other product at the abattoir premises⁸⁸ to the source premises
- the destination or intended use of the product (including the potential for exposure of poultry or other birds, or other animals. such as via pet food)
- biosecurity during transport of the product.

The movement of meat and meat products other than that derived from, or contaminated by, meat or meat products from an IP, DCP or SP is considered low risk in terms of likelihood of being contaminated prior to arriving at the abattoir, and low consequence as other controls (e.g. prohibited feeding of poultry products to poultry) will be in place.

Movement controls should be applied on a risk-assessed basis where:

- there is suspicion that an animal was infected when received by the abattoir, or
- the meat or meat products may have been cross-contaminated at the abattoir premises, or
- identification and tracing processes, including consideration to the date and time of processing, cannot preclude that the processed product was infected or the product was cross-contaminated by infective material.

All product that may have been contaminated is designated to the highest risk premises classification.

Table 6.3 addresses these considerations.

6.4.3.1 Management of product at an abattoir premises

The following is for management of animal product or by-product derived from poultry moving under permit (or moving under normal jurisdictional or interstate movement requirements for OA-to-OA movements), as well as product or byproduct that is held on site at an abattoir premises at the time it is classified as an IP, DCPF, APF, UPF, SP or TP.

⁸⁷ If test results are pending, it is possible that poultry or product suspected of being contaminated with AI may need to be destroyed and/or disposed of if it is impractical to hold product until test results are available.

⁸⁸ For the purposes of this manual, an abattoir premises is a premises where the abattoir is located. It may include additional structures on the same site such as chillers and cold storage facilities.

The following does not apply to animal product or byproducts that have moved off the abattoir premises at the time it is classified as an IP, DCPF, SP or TP.

Further restrictions on movement are unlikely once product is released into the retail market.

Table 6.2 Recommended movement controls for meat and meat products from abattoirs within and between areas

To → From ↓		RA/CA/OA
Area where abattoir located	Abattoir classification	
RA/CA	APF	Allowed under GP – conditions 4, 6, 7, 8
	DCPF	If poultry originated from the OA, a POR, ARP, TP, SP or DCP: prohibited (except under SpP – conditions 1, 3, 4, 5, 6, 7, 8)
		If poultry originated from an IP: prohibited (except under SpP – conditions 1, 2, 3, 7, 8)
	IP	If birds originated from an IP: prohibited (except under SpP – conditions 1, 2, 3, 7, 8)
If birds originated from the OA, a POR, ARP, SP, TP or DCP: prohibited (except under SpP – conditions 1, 3, 4, 5, 6, 7, 8)		
	SP, TP, UPF	Prohibited (except under SpP – conditions 1, 3, 4, 5, 6, 7, 8)
OA	Abattoir premises	If poultry originated from the OA, meat derived from those poultry is allowed to move under normal jurisdictional or interstate movement requirements
		If poultry originated from a POR or ARP: prohibited (except under GP conditions 3, 4, 6, 7, 8)

APF = approved processing facility; ARP = at-risk premises; CA = control area; DCP = dangerous contact premises; DCPF = dangerous contact processing facility; GP = general permit; IP = infected premises; OA = outside area; POR = premises of relevance; RA = restricted area; SP = suspect premises; SpP = special permit; TP = trace premises; UPF = unclassified processing facility

Permit conditions for Table 6.2:

1	Documented risk assessment that indicates that the risk associated with the meat or meat product movement is acceptable within the response.
2	For disposal or treatment (virus inactivation) that inactivates the AI virus.
3	Biosecure transport of meat or meat products.
4	Consigned poultry passed ante- and post-mortem inspection.
5	Consigned animals were not processed after poultry from an IP (farm) unless an appropriate decontamination process had occurred after poultry from the IP (farm) and prior to the consigned animals being processed.
6	Abattoir is verified by an abattoir biosecurity expert as operating in accordance with Sections 5, 8, 9, 10 and 20 of AS 4696:2023 <i>Australian standard for the hygienic production and transportation of meat and meat products for human consumption</i> ⁸⁹ to mitigate the likelihood of cross-contamination during processing.
7	The meat or meat product or packaging, including transportation equipment such as pallets/bins, is not brought into direct or indirect contact with susceptible animals.
8	The transport vehicle and driver are not brought into direct or indirect contact with susceptible animals or stock trucks unless there is no meat/meat product on board and the vehicle and driver have been decontaminated.

6.4.4 Eggs and egg products

Table 6.3 describes the recommended movement controls for all types of eggs, and egg products, within and between areas.

Egg products that have been treated in a manner that inactivates virus (e.g. pasteurisation) are excluded from the below requirements, provided that packaging and transport equipment that has been in contact with the product or its packaging is decontaminated upon leaving facilities that handle untreated products (e.g. a DCPF).

⁸⁹ <https://store.standards.org.au/product/as-4696-2023>

Table 6.3 Recommended movement controls for eggs and egg products within and between areas

ADS = approved disposal site; APF = approved processing facility; ARP = at-risk premises; CA = control area; DCP = dangerous contact premises; DCPF = dangerous contact processing facility; GP = general permit; IP = infected premises; OA = outside area; POR = premises of relevance; RA = restricted area; SP = suspect premises; SpP = special permit; TP = trace premises; UPF = unclassified processing facility

To→		RA									CA						OA
From↓		IP	DCP	SP	TP	DCPF ⁹⁰	ADS	APF	UPF ⁹¹	ARP	SP	TP	DCPF ⁹²	ADS	APF	UPF ⁸⁵	
RA	IP	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10)	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10)	Prohibited			Prohibited						Prohibited
	DCP	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10)	Prohibited			Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 11)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10)	Prohibited			Prohibited						Prohibited
	SP	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 11, 12)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10)	Prohibited			Prohibited						Prohibited

⁹⁰ For example, egg processing plant; grading facility

⁹¹ A UPF is an abattoir, knacker, milk or egg processing plant or other such facility where the current presence of susceptible animals and/or risk products, wastes or things is unknown. UPF can be used as a default status in a response until there is sufficient information to reclassify it. UPFs cannot receive live animals.

⁹² For example, egg processing plant

To→		RA								CA							OA	
From ↓		IP	DCP	SP	TP	DCPF ⁹⁰	ADS	APF	UPF ⁹¹	ARP	SP	TP	DCPF ⁹²	ADS	APF	UPF ⁸⁵		POR
RA	TP	Prohibited			Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 10, 13, 14, 15)	Prohibited	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited	Prohibited	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 9, 10, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15)	Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15)	Prohibited	Prohibited
	ARP	Prohibited			Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 10, 11, 12, 13, 14, 15)	Prohibited	Prohibited (except under GP – conditions 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited (except under GP – conditions 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited	Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)			Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)
	UPF	Prohibited			Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 15)			Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)		Prohibited	Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 15)		Prohibited	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 15)	Prohibited	Prohibited
	DCPF	Prohibited			Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 15)			Prohibited			Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 15)	Prohibited			Prohibited

To→		RA								CA							OA		
From ↓		IP	DCP	SP	TP	DCPF ⁹⁰	ADS	APF	UPF ⁹¹	ARP	SP	TP	DCPF ⁹²	ADS	APF	UPF ⁸⁵		POR	
RA	APF	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 15)	Not applicable	Prohibited (except under GP – conditions 2, 3, 4, 5, 6, 7, 8, 10, 11, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 15)	Prohibited	Prohibited	Prohibited	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 15)	Not applicable	Prohibited (except under GP – conditions 2, 3, 4, 5, 6, 7, 8, 9, 11, 15)			Prohibited	Prohibited (except under GP – conditions 2, 3, 6, 7)
CA	SP	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 8, 9, 11, 12)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10)	Prohibited			Prohibited	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 8, 9, 11, 12)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10)	Prohibited			Prohibited		
	TP	Prohibited			Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 13, 14, 15)	Prohibited			Prohibited	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10, 12, 13, 14, 15)	Prohibited			Prohibited		
	POR	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10)	Prohibited (except under GP – conditions 2, 3, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)	Prohibited	Prohibited (except under GP – conditions 2, 3, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)	Prohibited	Prohibited	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited	Prohibited (except under GP – conditions 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited (except under GP – conditions 1, 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited (except under GP – conditions 1, 2, 3, 5, 6, 7, 8, 11, 12, 13, 14, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)

To→		RA									CA							OA	
From ↓		IP	DCP	SP	TP	DCPF ⁹⁰	ADS	APF	UPF ⁹¹	ARP	SP	TP	DCPF ⁹²	ADS	APF	UPF ⁸⁵	POR		
CA	UPF	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 15)	Prohibited (except under GP – conditions 2, 3, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15)			Prohibited	Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 11, 12, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 12, 15)	Prohibited			Prohibited
	DCPF	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 15)	Prohibited			Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 11, 12, 15)	Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 12, 15)	Prohibited			Prohibited	
	APF	Prohibited				Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 15)	Not applicable	Prohibited (except under GP – conditions 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 15)			Prohibited	Prohibited		Prohibited (except under SpP – conditions 1, 2, 3, 4, 5, 6, 7, 8, 11, 12, 15)	Not applicable	Prohibited (except under GP – conditions 2, 3, 4, 5, 6, 7, 8, 11, 12, 15)	Prohibited		Allowed under normal jurisdictional requirements
OA	Prohibited						Allowed under normal jurisdictional requirements	Prohibited (except under GP – conditions 2, 3, 4, 5, 6, 7, 8, 9, 11, 12, 15)	Allowed under normal jurisdictional requirements	Prohibited			Allowed under normal jurisdictional requirements	Prohibited	Allowed under normal jurisdictional requirements	Allowed under normal jurisdictional and interstate movement requirements			

Permit conditions for Table 6.3:

1	A satisfactory risk assessment to determine whether the risk associated with the movement is acceptable within the response, including any conditions required to manage the product at the receiving premises.
2	Biosecurity practices, including decontamination, must be implemented for vehicles and equipment when entering and exiting properties to minimise the risk of disease spread. If items such as egg fillers or packaging cannot be decontaminated (e.g. cardboard egg fillers), they must not be used in the first instance, or must be new and clean on use, and disposed of at the destination.
3	Transport must be undertaken in a biosecure manner.
4	Transport by main roads/highways and not transiting through a property or stopping en route adjacent to a known poultry production area.
5	Transport vehicles are decontaminated at an appropriate site (e.g. truck wash-down facility) immediately after unloading and under supervision (government, industry or accredited third party). Decontamination must occur before entry to a new poultry premises or poultry product processing facility within the destination declared area or before leaving the destination declared area.
6	Any material permitted for movement must not be brought into direct or indirect contact with susceptible livestock.
7	The destination premises must implement biosecurity standards that minimise the risk of contaminated product contributing to viral spread, and must have mechanisms that minimise the likelihood of wild animals accessing the waste product material.
8	Material must be treated in a manner that meets requirements for inactivation of AI virus before onward movement of material to another destination.
9	Movement only considered where there is no destination facility in the same declared area.
10	Embryonated eggs must be destroyed before disposal. Refer to the AUSVETPLAN Operational manual: <i>Destruction of animals</i> .
11	Processor must process eggs such that they do not provide a risk for disease spread (e.g. egg surface decontamination, pulping, pasteurisation etc.).
12	Egg surfaces are decontaminated at source. If they cannot be decontaminated, they must be disposed of.
13	Absence of clinical signs consistent with AI in all poultry on the premises of origin.
14	Any suspicious or consistent clinical signs of AI in poultry are immediately reported to the relevant jurisdiction or through the Emergency Animal Disease Hotline (1800 675 888).
15	Entire consignment is from a single premises (no multiple pick-ups) and for delivery to a single premises (no multiple drop offs).

6.4.5 Waste products

Waste products are defined as manure, effluent, skins, feathers, blood, offal and other animal products not destined for human consumption.

Table 6.4 describes the recommended movement controls for waste products and effluent within and between declared areas.

Where movement of waste products is allowed, only direct movements for disposal/virus inactivation are permissible (as per permit condition 1, below) and the material must not be brought into direct or indirect contact with susceptible livestock (as per permit condition 7, below).

Table 6.4 Recommended movement controls for waste products and effluent within and between declared areas

To → From ↓		RA	CA	OA
RA	IP, SP, DCPF	Prohibited (except under SpP – conditions 1-7)	Prohibited (except under SpP – conditions 1-8)	Prohibited
	DCP, TP	Prohibited (except under SpP – conditions 1-7, 9, 10)	Prohibited (except under SpP – conditions 1-10)	Prohibited
	ARP, APF	Prohibited (except under GP – conditions 1, 3-7, 9, 10)	Prohibited (except under SpP – conditions 1-7, 9, 10)	Prohibited
CA	SP, DCPF	Prohibited (except under SpP – conditions 1-7)		Prohibited
	TP	Prohibited (except under SpP – conditions 1-7, 9, 10)		Prohibited
	POR, APF	Prohibited (except under GP – conditions 1, 3-7, 9, 10)		Prohibited (except under SpP – conditions 1-10)
OA		Allowed under normal jurisdictional requirements		

Permit conditions for Table 6.4:

1	Direct movement from premises of origin to approved disposal site or premises/processing facility that treats the product in a manner that meets requirements for inactivation of AI virus e.g. rendering.
2	A satisfactory risk assessment to determine whether the risk associated with the movement is acceptable within the response, including any conditions required to manage the product at the receiving premises.
3	Biosecurity practices, including decontamination, must be implemented for vehicles and equipment when entering and exiting properties known or likely to be contaminated, to minimise the risk of disease spread. Transport vehicles must be decontaminated (i.e. cleaned and disinfected) at an appropriate site (e.g. truck wash-down facility) immediately after unloading and under supervision (government, industry, or accredited third party). Decontamination must occur prior to entering a new poultry premises or poultry product processing facility within the destination declared area, or before exiting the destination declared area.
4	Transport must be undertaken in a biosecure manner and in leakproof trucks, vehicle trays or containers.
5	Transport by main roads/highways and not transiting through a property or stopping en route adjacent to a known poultry production area.
6	Any material permitted for movement must not be brought into direct or indirect contact with susceptible livestock.
7	The receiving premises must implement biosecurity standards that minimise the risk of contaminated product contributing to viral spread, and must have mechanisms that minimise the likelihood of wild/feral animals accessing the waste product material.
8	Movement only considered where there is no disposal or other relevant processing facility in the same declared area.
9	Absence of clinical signs consistent with AI in animals on the premises before and on the day of dispatch.
10	Any clinical signs in animals suspicious for, or consistent with, AI are immediately reported to the local control centre, state coordination centre or Emergency Animal Disease Hotline (1800 675 888).

6.4.6 Vehicles and equipment

This includes empty livestock transport vehicles and associated equipment.

6.4.6.1 Movements of vehicles and equipment that have had direct contact with susceptible animals, or their products or wastes (including with potentially contaminated soil, etc.)

- Movements onto and off quarantined premises (i.e. IPs, SPs, TPs, DCPs, DCPFs and ADSs) are prohibited except under SpP and subject to risk assessment on a case-by-case basis. Where movements are permitted under SpP, the vehicles and equipment must be decontaminated before and after use at an appropriate site using a protocol provided by the response authority under the supervision of an authorised officer. Where decontamination of equipment is not practicable, the equipment should be disposed of in a biosecure manner. Records must be kept of the movement and decontamination protocol used.
- Movements onto and off other premises not under quarantine (premises other than IPs, SPs, TPs, DCPs, DCPFs and ADSs) in the RA are prohibited except under GP. Where movements are permitted under GP, the vehicles and equipment must be decontaminated before and after use at an appropriate site using a protocol provided by the response authority. Where decontamination of equipment is not practicable, the equipment should be disposed of in a biosecure manner. Records must be kept of the movement and decontamination protocol used.
- Movements onto and off of premises not under quarantine (premises other than IPs, SPs, TPs, DCPs, DCPFs and ADSs) in the CA are prohibited except under GP, with the conditions that the vehicles and equipment must be decontaminated before and after use at an appropriate site (e.g. truck wash-down facility at an abattoir) using a protocol provided by the response authority, and records must be kept of the movement and decontamination protocol used.
- Movements off premises in the OA are not restricted.

6.4.6.2 Movements of other vehicles and equipment

- Movements onto or off quarantined premises (IPs, SPs, TPs, DCPs, DCPFs and ADSs) are restricted and subject to risk assessment on a case-by-case basis.
- Movements onto or off other premises with susceptible animals in the RA or CA should be restricted to essential movements only. Regular, routine vehicle movements onto farms, such as those for feed deliveries, require particular attention because of the essential nature of these movements, their frequency and the risk that they may present. Removal of vehicle and equipment gross contamination should be undertaken when observed, and periodic full decontamination of vehicles and equipment as a precautionary measure should be undertaken at the frequency nominated by the response authority. Records must be kept of the movement and decontamination protocol used.
- Movements onto or off other premises in the OA are not restricted.

6.4.7 Nonsusceptible animals

Nonsusceptible animals should be prevented from coming into proximity with poultry facilities. However, where this has already occurred and where nonsusceptible animals could act as a transmission pathway from high-risk premises (i.e. IPs, DCPs, DCPFs, SPs, TPs and ADSs), appropriate decontamination measures should be implemented before they are moved from the premises.

Unnecessary movements of nonsusceptible animals onto and off premises with susceptible animals in RAs should be discouraged.

Movements of nonsusceptible animals in the CA and OA should not be restricted.

6.4.8 People

Movement controls should not hinder movements of the general public on non-quarantined or non-infected areas.⁹³ However, where humans could act as mechanical vectors for AI virus – for example, on IPs, DCPs, DCPFs, SPs, TPs and ADSs – appropriate personal protection (see also Section 4.2) and personal decontamination measures should be implemented.

Should human infection with the outbreak virus occur, human health authorities will manage public health issues.

Unnecessary movements of people onto and off premises with susceptible animals in the RA should be discouraged.

Within the RA, people who regularly travel from farm to farm and come into contact with susceptible animals will be required to undergo appropriate decontamination of themselves, and their outer wear, equipment and vehicles between properties, and keep detailed records of their movements. They will be required to follow biosecurity controls at each premises they visit.

People involved in delivering feed and other essential materials (e.g. water, gas, diesel) to quarantined premises, including IPs, DCPs, DCPFs, SPs, TPs and ADSs, should comply with the following conditions:

- driver should not exit the cabin of the truck (where practicable)
- driver should not contact poultry
- driver and cab are decontaminated if driver exits the truck on the premises (both cab and driver are decontaminated before the driver re-enters the cab).⁹⁴

Movements on premises other than SPs and TPs within the CA and OA will not be restricted.

⁹³ There may be restrictions placed on people movement on infected areas to control exposure to AI virus infected wildlife.

⁹⁴ Vehicle requirements are as per Section 6.4.6.

6.4.9 Crops, grains, hay, silage and mixed feeds

Crops, grains, hay and silage harvested from paddocks that were sprayed or treated with effluent on an IP, DCP or SP, or mixed feeds made from such constituents, are not permitted to be moved off-site until the premises is declared free from AI and appropriate decontamination has occurred.⁹⁵ Other crops and grains may be removed from IPs and DCPs after the material has been decontaminated, and moved to other premises in either the RA or the CA, provided that the people, vehicle and equipment movement requirements are observed. Crops and grains may be moved from TPs following risk assessment on a case-by-case basis and upon meeting movement permit conditions.

Movements of feeds onto IPs, DCPs, SPs and TPs may be necessary for animal welfare reasons; these would be permitted from low-risk premises or premises in the OA, provided that the people, vehicle and equipment movement requirements are observed.

Crops and grains from premises not associated with an IP, DCP, SP or TP have no movement restrictions.

Other feed movements that have, or may have, an association with an IP, DCP, SP or TP will be risk assessed on a case-by-case basis.

Risk management of stored feed on HPAI or LPAI (H5/H7) properties

In assessing the potential disease risk from feed stored in silos or trucks on a feedmill premises associated with IPs, DCPs, SPs or TPs, the likelihood of contamination of feed must be considered.

Sources of contamination include:

- dust
- aerosols
- movement of infected or potentially infected birds from sheds, resulting in virus aerosolisation, or windborne spread of dust or feathers
- eggs (potential for virus transmission via direct or indirect contact)
- handling or removal of manure or litter, resulting in windborne spread of dust
- contamination from fomites
- human movements – cross-contamination can be minimised by biosecurity practices, including the use of dedicated staff for various elements of the farm (e.g. poultry sheds, feedmill operations)
- mechanical transmission by animals (e.g. dogs, cats, rodents, wild birds) or insects (e.g. flies), or contamination by infected animals (wild birds).

In assessing the potential disease risk from feed stored in silos or trucks on IPs, DCPs or SPs (but not in a feedmill), the likelihood of feed contamination by the following means must be considered:

- feed delivered from the feedmill that is already contaminated with AI virus through mechanisms described above
- introduction of virus into silos or trucks during loading of the feed, where the virus source is from items noted above
- introduction of virus following loading of silos or trucks that are not fully sealed.

Storage conditions (e.g. time, temperature, location, security) and treatments (e.g. fumigation, pelleting, acidifying) will affect the viability of the virus in stored contaminated feed.

The impact of spread of AI virus via contaminated feed should also be considered, taking into account the proposed use (e.g. feeding to poultry on other farms or restocked populations; feeding to other susceptible or potentially susceptible species) or fate (e.g. disposal) of the feed. Where the feed is to be disposed of,

⁹⁵ This will be informed through risk assessment, taking into account environmental conditions, including ambient temperature and humidity.

consideration must be given to disposal procedures, and time and exposure pathways that may be created during disposal; for example, feed to be buried that is left uncovered may allow wild bird access.

6.4.10 Sales, shows and other events

Sales, mobile petting zoos, shows or exhibitions, and other events involving live susceptible animals:

- within the RA and CA are prohibited
- in the OA may proceed at the discretion of the relevant jurisdictional CVO, unless the risk associated with such events is deemed unacceptable within the response.

6.4.11 Other movements

Other susceptible species (e.g. birds and susceptible non-human mammals) should not be moved from IPs, DCPs, DCPFs, SPs, TPs and ADSs without risk assessment, appropriate permit and conditions.

7 Surveillance, restocking and proof of freedom

Following an outbreak of avian influenza (AI), surveillance will be required to demonstrate that infection has been eradicated from the commercial poultry population.

This surveillance can be considered in 2 parts:

- surveillance on premises to assist with assessment or resolution of premises classifications and declared areas thereby enabling remaining movement controls to be lifted (i.e. 'surveillance on premises following response activities'). This includes the use of sentinel birds.
- surveillance to provide evidence of absence of infection of HPAI in commercial poultry to enable a WOAHP self-declaration of freedom from HPAI (i.e. 'proof of freedom surveillance').

The **AUSVETPLAN Guidance document: *Declared areas and allocation of premises definitions in an EAD response*** provides guidance on reclassifying previously declared areas, including surveillance activities that support the resolution.

The WOAHP *Terrestrial animal health code* includes recommended criteria for providing a self-declaration of freedom from HPAI in poultry (as defined in the code); however, there are no WOAHP recommendations to provide evidence of freedom from LPAI H5/H7.

Evidence of proof of freedom surveillance will also be needed to satisfy trading partners and regain access to international markets, noting some markets may be regained following individual negotiations during and following an outbreak, before a self-declaration of freedom is made.

A key requirement for trading partners will be evidence of an effective surveillance program capable of detecting infection if present in the population, and analysis of data to support the case for disease freedom. Descriptions of the veterinary services, demographics of relevant susceptible populations and relevant industry structures should be included to justify the design of the surveillance program.

7.1 Surveillance of sentinel birds on premises following response activities

7.1.1 Use of sentinel birds

Use of identifiable sentinel birds (see also Section 4.3.16) to confirm successful decontamination on AI virus contaminated premises is a valuable step in demonstrating freedom from AI.

7.1.1.1 Considerations

7.1.1.1.1 Species

Ideally, the sentinel bird species should be one that is highly susceptible to AI virus (especially to the outbreak strain). Chickens, turkeys, and other gallinaceous poultry are highly susceptible to clinical infection and would be the first choice. However, where consideration is being given to partial restocking as part of the sentinel process, other species could be used.

7.1.1.1.2 Age

Sentinel birds should be a minimum of 7 weeks of age, where practicable. Younger birds (e.g. day-old chicks) may be considered if using sentinel birds as part of a restocked broiler population so long as appropriate samples (e.g. swabs and blood) can be taken for post-placement sampling. Pre-placement testing of the parent flock should be considered where placed birds are too young for testing.

7.1.1.1.3 Health status

Consideration should also be given to the health status of the source flock and potential for introduction of endemic disease as part of the sentinel program.

7.1.1.1.4 Animal welfare

Selection of poultry suitable for sentinel bird placement should also consider other factors such as familiarity with the production system into which they will be placed to minimise compromised welfare. For example, birds that have been raised in a single-level barn system may not be suitable for placement into an aviary barn with raised feed, water and nests. In caged housing systems, placement of a single bird into cages for sentinel programs can be stressful and lead to poor welfare outcomes.

Changes to husbandry and management practices can lead to behavioural issues and poor welfare among poultry. Response activities such as the placement of sentinel birds into new production systems may lead to severe vent pecking and cannibalism within some flocks. Such behavioural issues are difficult to eradicate once they become established within a flock, and mitigation measures should be implemented to reduce the welfare risk.

7.1.1.1.5 Building or range access

It is essential that sentinel birds are exposed to areas that may have a risk of residual AI virus. For example, having sentinel birds roaming the floor of cage facilities is more desirable than having them caged. However, consideration must be given to the appropriateness of such action, for example bird access to food and water. In free-range facilities, sentinel birds must be allowed the option to roam all areas of the range production area.

Considerations on whether sentinel birds will be granted range access should take into account the longer-term restocking plan (e.g. restocking with pre-point-of-lay pullets that would otherwise not have range access until after point-of-lay, or reconsidering whether they will be a free-range flock at all), implications of range access to young sentinel birds (e.g. industry practice is to now allow range access after point of lay), ability to decontaminate the range areas and known virus survival in various media (e.g. water, slurry) and ability to restock the range with enough sentinels to be adequately exposed to any remnant viable virus within the sentinel program. If free-range properties do not allow sentinel birds access to the range, jurisdictions will need to decide if this affects the time until commercial use of the range is permitted following risk assessment of virus survivability in the environment.

7.1.1.1.6 Numbers of sentinels

The number of sentinel birds should include an allowance (3-5%) for losses unrelated to AI that may occur over the 21–28 day observation period. This will ensure that enough birds are available for testing at the end of the period.

If sentinel birds show clinical signs following pre-placement sampling that could indicate AI, further investigation must be undertaken, and birds must test negative for AI before the birds are used as sentinels. Alternatively, substitute birds could be sourced.

7.1.1.2 Testing

Testing should be undertaken at the source property of the sentinel birds less than 7 days prior to placement (except in cases where day-old chickens are used). Cloacal and tracheal swabs and serum should be tested by polymerase chain reaction (PCR) and serology respectively, to demonstrate absence of infection prior to sentinel bird placement on the premises. It is important to consider the potential for false positive results and have a strategy in place to resolve these if they occur.

If repopulating takes more than 7 days, on the last day of repopulating, birds on the source property are also to be sampled using an epidemiologically representative sample size. This will ensure that if the premises breaks down, it is not due to contamination from the source property.

A pre-movement (within 24-hours of movement) clinical inspection is also required. If sentinel birds show clinical signs following pre-placement sampling that could indicate avian influenza, appropriate samples should be taken and test negative for avian influenza before the birds are used as sentinels. Alternatively, substitute birds should be sourced.

Cloacal and tracheal swabs (PCR) and serum (serology) should be collected a minimum of 21–28 days from sentinel birds after introduction to the premises. Sample sizes are provided in Table 7.1.

Table 7.1 Sample sizes for testing of sentinel birds^{a b}

Barn/litter birds		Caged birds	
Population per shed	Sample size per shed	Population per shed	Sample size per shed
≤30	All	≤40	All
31–60	31	41–60	41
61–100	33	61–100	55
101–200	35	101–200	60
>200	40	>200	64

^a Assumptions for barn/litter birds – sensitivity 98%, specificity 98%, design prevalence 15%; assumptions for caged birds – sensitivity 98%, specificity 98%, design prevalence 10%.

See also: Sergeant (2018). Epitools epidemiological calculators (<https://epitools.ausvet.com.au>).

^b Sample size calculations are based on individual samples. If pooling of samples is proposed, sample size calculations should be verified.

Daily health monitoring must be completed for as long as sentinel birds are in place and all dead birds must be tested for evidence of avian influenza infection.

Sentinel birds may not need to be destroyed at the end of the sentinel period. Depending on the numbers involved, and the species and age of the birds, if they exhibit no clinical, serological or virological evidence of AI infection, they may be reclassified from sentinel to commercial and become part of staged restocking.

7.2 Restocking

7.2.1 Principles

No restocking should take place before the outbreak has been brought under control.

Restocking of flocks or areas may be undertaken after a risk assessment and consideration of the epidemiological situation on the premises and in the declared areas.

When determining the time between completion of destruction and restocking of premises with sentinels or commercial restocking (i.e. the empty period), virus viability outside the host should be considered. Factors influencing virus viability include the decontamination method used, the virus type, temperature, humidity, salinity, pH, surface type and ultraviolet light (see also Appendix 3). Consideration should also be given to the enterprise type (e.g. barn vs free-range). During the empty period, poultry shed(s) need to be secured so that the site cannot be re-contaminated by wild or domestic birds. The minimum empty period for free-range situations may be longer because of the challenges of decontaminating the range.

Tracheal and cloacal swabs and serum for PCR and serology will be taken from an appropriate sample size (see Table 7.1) a minimum of 21–28 days after placing sentinels or partially or completely repopulating the premises. During this time, any unusual health incidents are to be fully investigated, including testing of samples for evidence of AI infection.

Dead bird sampling of either sentinels or restocked birds should be undertaken during the 21–28 day observation period to confirm the ongoing absence of AI.

Daily health monitoring should be completed for a minimum of 21–28 days, with investigation of any birds showing clinical signs that could indicate AI.

To provide assurance of disease freedom, cloacal and tracheal swabs (PCR) and serological sampling should be undertaken at a minimum of 21–28 days post-placement of sentinels or restocked birds.

Section 2.4.2.2 and Appendix 3 provide further details on virus viability under a range of environmental conditions that will help inform the duration of the empty period.

7.2.2 Restocking options

When restocking, the following 2 options can be considered:

- commercial restocking without sentinels
- sentinels only, prior to commercial restocking.

The sample sizes to test are as per Table 7.1 for all options (plus extra birds – eg 3–5% of the sample size – to account for any mortality).

7.2.2.1 Commercial restocking without sentinels

Where deemed appropriate, based on the outcome of risk assessment, commercial restocking may be undertaken without the use of sentinels.

Historically in Australia, full restocking has not been allowed until at least 14 days following decontamination. Full restocking without sentinels is subject to risk assessment in accordance with the principles in Section 7.2.1.

Prior to allowing commercial restocking of a property with an IP status, consideration should be given to the management of the eggs and waste produced while the property undergoes final resolution. Surveillance requirements of birds (pre- and post-placement) should also be considered.

The health status of birds to be used for restocking should be assessed prior to placement. This may involve assessing the health status of parent flocks and/or replacement stock prior to placement (PCR and serology), plus clinical inspection 24 hours pre-placement/movement. Pre-placement testing of commercial numbers of birds has been shown to produce false positive results (especially serology) and therefore planning must consider how these should be managed.

7.2.2.2 Sentinels only, prior to commercial restocking

Placement of sentinels prior to commercial restocking may be undertaken following decontamination and the empty period.

The maximum stocking density is 30 kg/m² for non-caged layers or pullets. In a barn or litter shed, the sentinels are allowed to roam on the floor. For testing, the sentinels are sampled, with no requirement to retest the same birds.

For free-range facilities, sufficient numbers should be used to allow the birds to be exposed to all areas potentially contaminated with AI virus.

In a caged bird shed, the sentinels would ideally roam on the floor. If it is not logistically possible to provide accessible food and water to the birds with this setup, they are instead placed randomly throughout the shed, with their cages identified.

7.2.3 Surveillance in declared areas and the outside area

The following surveillance activities may be considered for demonstrating the absence of AI. It has been successfully used in previous high pathogenicity avian influenza (HPAI) outbreaks in New South Wales (Maitland in 2012 and Young in 2013–14).⁹⁶

7.2.3.1 Surveillance in the restricted area and control area

Surveillance to be undertaken in the restricted area (RA) and the control area (CA) may include:

- identification and mapping of all commercial poultry premises in the RA and CA; each owner is to be advised by the local control centre (LCC) to immediately report health issues to the state or territory department of agriculture and arrange:
 - daily health monitoring⁹⁷, with a daily report to be submitted to the LCC
 - weekly sampling of flocks (cloacal swabs for PCR, preferentially sampling dead birds) on each commercial poultry premises, with final sampling 21-28 days after depopulation on all infected premises (IPs)
- prompt investigation of any reports of disease in non-commercial poultry flocks, captive birds, or wild birds to support a passive surveillance program for non-commercial domestic avian species.

⁹⁶ Information provided by the New South Wales Department of Primary Industries.

⁹⁷ Health monitoring includes identifying and reporting; a decline in feed and/or water consumption; mortality rates and the relationship of daily mortality figures to normal figures; production rates and any decline in egg production from normal; any clinical signs attributable to AI; a statement declaring the health or otherwise of the poultry.

A communications plan should be implemented targeting owners of non-commercial poultry and captive birds in the RA and CA advising of the need to report bird health issues to the relevant jurisdictional authority. Consideration may be given to the identification and mapping of non-commercial poultry flocks and captive birds.

7.2.3.2 Surveillance in the outside area

In the outside area, poultry producers, veterinarians and members of the public are requested to report any suspicion of AI to the relevant state or territory department of agriculture as soon as possible.

7.2.3.3 Removal of declared areas

Declared areas may be reclassified or revoked when:

- all TPs and SPs have been reclassified, and all IPs, DCPs and DCPFs in the area have been reclassified as resolved premises (RPs)
- all tracing and surveillance associated with AI control measures has been completed satisfactorily, with no evidence or suspicion of infection in the area
- a minimum period of 28 days has elapsed since disease control activities and risk assessments were completed on the last IP or DCP in the area
- an approved surveillance program (including the use of sentinel animals, if appropriate) has confirmed no evidence of infection in the RA.

See also the **AUSVETPLAN Guidance document: *Declared areas and allocation of premises definitions in an EAD response***. on reclassifying previously declared areas.

7.2.3.4 Surveillance following removal of restricted and control areas

Surveillance in the 3 months following removal of the RA and CA should consist of passive surveillance in both previously infected and uninfected areas, including:

- review of laboratory submissions and reports that have features consistent with AI
- investigation of all suspect cases (reports of disease consistent with AI) in poultry and wild birds
- analysis of reports from routine wild bird surveillance in jurisdictions where wild bird surveillance projects are in place.

Active surveillance during this period should also include ongoing health monitoring on RPs. Jurisdictional departments of agriculture should be notified of the presence or absence of clinical disease on these premises.

7.3 Proof of freedom

The WOAH *Terrestrial animal health code* (Article 10.4.3) lists the criteria (see also Section 4.1.6) for a previously AI-free country or zone to be considered free from infection with HPAI viruses in poultry following an HPAI outbreak (note that the WOAH definition of poultry will apply).

To underpin an official self-declaration of AI-free status following an outbreak, Australia would develop a formal report detailing the eradication procedures undertaken, the surveillance program and the results reported. This report could be provided to trading partners to transparently document the basis for our self-declaration.

Trading partner acceptance of AI-free status following an outbreak will most likely have to be negotiated with individual trading partners. This may take considerably longer than the minimum periods prescribed in the WOAH *Terrestrial animal health code*.

7.3.1 Principles for designing a proof of freedom surveillance program

To provide confidence that AI is no longer present, a comprehensive surveillance program will be required, and evidence of this program may need to be provided to international trading partners to support export market access. This program will need to be carefully designed, detailed and followed to ensure that it produces sufficient data that are reliable and acceptable to international trading partners, while avoiding being excessively costly and logistically complicated.

Following an outbreak, the program must demonstrate absence of infection with AI viruses in susceptible poultry populations during the 28 days following decontamination of the last infected premises as per the WOAH *Terrestrial animal health code* Article 10.4.6. The proof of freedom surveillance program will build on the previous surveillance, tracing and diagnostic testing done during the response phase. It should include clinical and serological surveillance, and targeted and random components.

7.3.1.1 Passive clinical surveillance

The aim of passive clinical surveillance is to look for evidence of infection by detecting clinical signs of AI at the flock level using physical examination of susceptible animals. It involves monitoring of production parameters – such as increased mortality, reduced feed and water consumption, reduced production rates and/or a decline in egg production – and checking for the presence of clinical signs consistent with a respiratory infection with AI virus. When clinical disease is suspected, laboratory testing should be undertaken by sampling and testing animals suspected of infection. This may include serology and virus isolation.

The approaches used for passive clinical surveillance will be a continuation of measures in place during the response and should include:

- a public relations and awareness campaign telling poultry producers (including backyard producers), zoos, captive bird owners, company technical personnel and animal health professionals (e.g. veterinarians, stock inspectors, meat inspectors) to immediately report suspicions of AI to government veterinary services
- effective veterinary investigations and diagnostic services that demonstrate that suspect cases are promptly investigated, ensuring notification of suspected disease to authorities on suspect premises pending diagnosis
- enhanced clinical inspection of poultry at abattoirs
- use of a standardised investigation protocol and reporting.

7.3.1.2 Active surveillance

In addition to clinical and/or laboratory investigation of suspect cases reported to authorities (passive surveillance), some active surveillance would also be expected to look for the disease in groups of animals that are at particularly high risk. This may include premises with domestic waterfowl production, multi-age flocks, premises that use untreated surface water as a drinking water source for birds, and premises with more than one bird species on the premises.

7.3.1.2.1 Sampling regime

Surveys based on random sampling are important in providing reliable evidence that AI virus infection is not present in 'poultry' (WOAH definition) in a country. The sampling strategy will be designed to demonstrate the absence of AI virus circulation at an acceptable level of statistical confidence. Important factors that must be considered when designing the sampling regime include:

- design prevalence – the minimum level of infection that would be detected if the disease is present
- target population – the population under surveillance, which should cover all susceptible species, including, if appropriate, wild birds
- level of statistical confidence required in the results
- sensitivity and specificity of diagnostic tests
- sample size – number of flocks to be sampled and number of birds to be sampled per flock.

Attention must be paid to selecting an appropriate design prevalence and statistical confidence level for surveys, because these parameters will have to be justified and withstand international scrutiny. No diagnostic tests are perfect, so the survey design should anticipate the occurrence of false positive reactions and incorporate appropriate follow-up procedures.

7.3.1.3 Wild bird surveillance

Surveillance of wild bird populations will be commensurate with the level of assessed risk posed to domestic bird and wild bird populations and the potential for circulation of AI virus between domestic and wild bird populations.

Mortality events, or clusters of birds found dead should be reported and investigated, including testing of appropriate samples.

Active surveillance, may be necessary for detection of some strains of AI viruses that produce infection without mortality in wild birds.

Further information is provided in the WOA *Terrestrial animal health code* (Article 10.4.29).

7.3.1.4 Monitoring of low pathogenicity avian influenza in poultry populations

A monitoring system should be in place to detect LPAI, however there are no WOAH requirements for notification. The monitoring system should aim to detect infected flocks, as infected flocks increase the risk of virus mutation to HPAI if they go undetected and managed.

Further information is provided in the WOA *Terrestrial animal health code* Article 10.4.30.

Appendix 1 Avian influenza fact sheet

Disease and cause

Avian influenza (AI) is caused by a number of influenza viruses, of which only influenza A viruses have been isolated from avian species; influenza viruses of the B, C and D types have never been isolated from birds.

AI viruses are classified into 2 pathotypes – high pathogenicity AI (HPAI) and low pathogenicity AI (LPAI) – based on either the lethality of the virus in experimentally inoculated chickens or molecular characteristics.

Species affected

Influenza A viruses have been isolated from most major bird families — at least 485 bird species, of which 258 are newly-affected since 2021. Experimentally, AI virus can infect almost all commercial, domestic and wild avian species.

Detections have also occurred in a wide range of non-human mammals and several livestock species, especially since the emergence of H5Nx 2.3.4.4b.

Humans are susceptible to infection with AI viruses.

Distribution

AI is widely distributed throughout the world, and outbreaks have occurred in Australia. Wild birds in Australia have been shown to actively carry and shed LPAI virus and are likely to be susceptible to HxNx HPAI viruses.

Potential pathways of introduction or evolution

Strains of HPAI virus could be introduced into Australia or evolve in Australia through:

- migratory birds and other natural non-migratory movements of infected wildlife, including but not limited to wild birds
- illegal importation of contaminated goods
- importation of contaminated poultry products, fomites, inanimate objects or people
- evolution from domestically circulating LPAI viruses.

Key signs

Key signs in chickens and turkeys include severe respiratory signs with excessively watery eyes and sinusitis; cyanosis of the combs, wattle and shanks; oedema of the head; ruffled feathers; loss of appetite; diarrhoea; nervous signs; and sudden death.

Eggs may be misshapen. The last eggs laid after the onset of illness frequently have no shells. Some severely affected hens may recover but rarely come back into lay.

Wild birds infected with HPAI may show a range of clinical signs, from being non-clinical to presenting with clinical signs similar to those seen in infected poultry. Wild birds infected with LPAI viruses usually show no clinical signs.

Spread

Outbreaks of HPAI from viruses of Australian origin, are understood to originate from wild birds that carry LPAI. LPAI viruses can come into contact with poultry via wild birds contaminating range areas, water or

food supplies. In these instances, in order for an HPAI outbreak to occur, the LPAI virus is required to evolve which can occur in poultry flocks, particularly when kept at high concentration. The infection can subsequently spread via movements of infected live birds, or faecally contaminated feed, vehicles, equipment, materials, clothing and footwear. Aerosol may also contribute to disease spread. In the 2024 New South Wales HPAI (H7N8) outbreak in the Hawkesbury Valley it is likely that aerosol spread of virus up to 2.4km from the index case resulted in transmission to other premises.

Overseas it has been demonstrated that H5Nx 2.3.4.4b can spread in the high pathogenicity form direct from wild birds to poultry and other birds, which is unlike all experiences with HPAI in Australia to date. In this case the risk of multiple incursions of HPAI in not just commercial poultry, but backyard poultry and other domestic birds is heightened.

Persistence of the virus

Influenza viruses are lipid-enveloped RNA viruses. Stability of AI viruses varies by subtype. In general, influenza viruses are fragile; however, some subtypes can persist longer under optimal conditions. The virus is stable over a pH range of 5.0–8.2.

Impacts for Australia

One of the biggest impacts of an outbreak of AI involving the poultry industry would be on the domestic economy. Chicken meat and eggs are produced very efficiently in Australia and provide the cheapest source of animal-based protein available to the population; this is reflected in the consumption rate. Loss of availability of these products would cause economic stress to the majority of the population and the domestic economy.

H5Nx clade 2.3.4.4b outbreaks overseas in wild bird species of high conservation status have had negative impacts on decades of conservation efforts, threatening endangered species, biodiversity and overall ecosystem health.

Appendix 2 Infective dose and virus shedding

Infective dose

The infective dose for avian influenza (AI) virus under experimental conditions, for infection of chickens, turkeys, ducks, geese and quail, depends on the species of origin of the virus isolate, the virus subtype, the species infected, the age of individuals and the route of infection. Examples from studies of infective doses are shown in Table A2.1.

Table A2.1 Experimental infective dose of avian influenza virus by species

Species	Infective dose	
	LPAI (EID ₅₀)	HPAI (EID ₅₀)
Domestic duck	10 ^{1.9} –10 ^{3.3}	–
White leghorn chicken	10 ^{5.8} –10 ^{7.7}	10 ^{1.2} –10 ^{4.7} (all virus origins) 10 ^{2.8} –10 ^{4.7} (turkey origin) 10 ^{1.2} –10 ^{3.0} (chicken origin)
Turkey	10 ^{4.0} –10 ^{6.0}	–
Japanese quail	10 ^{1.5} –10 ^{5.4}	–
Domestic goose	10 ^{1.7} –10 ^{5.6}	–

– = no data; EID₅₀ = median egg infective dose; HPAI = high pathogenicity avian influenza; LPAI = low pathogenicity avian influenza
Source: Swayne & Slemons (2008)

Virus shedding

Virions are released from tissues within 16 hours after initial exposure. Virus shedding occurs before the onset of clinical signs and may last longer than apparent disease. The infectious period extends from the time the virus is first detected in the bird to the time it is no longer detected in oropharyngeal or cloacal swabs. Examples of concentrations of virus that may be detected in respiratory secretions or faeces are given in Table A2.2.

Table A2.2 Concentrations of avian influenza virus detected in respiratory secretions or faeces

	Virus concentration	
	Respiratory secretions (EID ₅₀ /mL)	Faeces (EID ₅₀ /g)
LPAI	10 ^{1.1} –10 ^{5.5}	10 ^{1.0} –10 ^{4.3}
HPAI	10 ^{4.2} –10 ^{7.7}	10 ^{2.5} –10 ^{4.5}

EID₅₀ log 10 = median egg infective dose; HPAI = high pathogenicity avian influenza; LPAI = low pathogenicity avian influenza
Source: (Swayne & Beck 2005, Swayne et al 2000)

Viruses with the potential to be highly pathogenic for chickens and turkeys can be carried by birds, and shed in faeces and from the respiratory tract for at least 14 days and up to 30 days after clinical recovery from the disease. Cloacal shedding can continue for longer than 30 days after infection in the presence of immunosuppressive diseases or other physical stresses (Swayne et al 2020).

Experimentally infected ducks have been shown to shed HPAI (H5N1) virus via both the cloaca and the respiratory tract for at least 17 days (Hulse-Post et al 2005).

In pheasants, partridges and guineafowl, virus was able to be isolated for up to 7 days after infection during the outbreak in the USA in 1983–84 (Pearson et al 2003).

Experimentally infected cats excreted H5N1 virus from the pharynx and nose, and in faeces for at least 7 days. Pharyngeal swabs contained $10^{4.5}$ tissue culture infective dose (TCID)₅₀/mL, and nasal swabs $10^{2.5}$ – $10^{5.0}$ TCID₅₀/mL; virus titres in rectal swabs varied widely, but were of similar magnitude. This study suggested cat-to-cat transmission of H5N1 virus could occur through infected faeces (Rimmelzwaan et al 2006).

Appendix 3 Virus viability

Blank cells indicate information was not provided in the reference.

Table A3.1 Summary of available scientific literature on avian influenza virus viability

Temp (°C)	Duration of infectivity	Species / substrate	pH	AI virus strain / pathogenicity	Additional conditions	Reference
Faeces						
0	52-75 days ^a	Duck faeces		LPAI-H4N6, H5N1, H6N8		Nazir et al 2011
4	>14 days	Duck faeces	7.8	LPAI H6N2		Zarkov et al 2013
4	>32 days	Duck faeces	7.68	LPAI H7N2, H3N6		Webster et al 1978
4	8 weeks	Poultry faeces		H5N1	moist	Kurmi et al 2013
4	7 weeks	Poultry faeces		H5N1	dry	Kurmi et al 2013
4	35 days	Poultry faeces		H5N2	moist	Beard et al 1984
4	9 days	Poultry faeces		H5N2	dry	Beard et al 1984
4	>23 days	Chicken faeces		H7N2 (considered LPAI)		Lu et al 2003
5	7 weeks ^a	Duck slurry	6.1-7.3	H5N8	no lime	Schmitz et al 2020
5	<1 week	Duck slurry	10-12	H5N8, H5N9	treated with lime	Schmitz et al 2020
10	14-21 days ^a	Duck faeces		LPAI-H4N6, H5N1, H6N8		Nazir et al 2011
10	14-21 days	Duck faeces		LPAI H7N2, H3N6		Webster et al 1978
15	>6 days	Duck faeces	7.8	LPAI H6N2		Zarkov et al 2013
15-20	2-23 days	Chicken faeces		H7N2(considered LPAI)		Lu et al 2003
19-22.5	>96 hours	Chicken faeces		H5N8,	8.5	Hauck et al 2017
19-22.5	<24 hours	Chicken faeces		LPAI H6N2	8.5	Hauck et al 2017
20	4-7 days ^a	Duck faeces		LPAI-H4N6, H5N1, H6N8		Nazir et al 2011
22	>2 days	Duck faeces	7.8	LPAI H6N2		Zarkov et al 2013
22	>8 days	Duck faeces	7.68	LPAI H7N2, H3N6		Webster et al 1978
24	5 days	Chicken faeces		H5N1	dry and moist	Kurmi et al 2013
25	< 1 day	Chicken faeces	8.2	H5N1		Chumpolbanchorn et al 2006
25	2 days	Poultry faeces		H5N2	moist	Beard et al 1984
25	1 day	Poultry faeces		H5N2	dry	Beard et al 1984
25-32	4 days	Chicken faeces		H5N1	shade	Songserm et al 2006
28-30	12-36 hours	Chicken faeces		H7N2(considered LPAI)		Lu et al 2003
30	12-36 hours	Chicken faeces		H7N2(considered LPAI)		Lu et al 2003
30	2 days ^a	Duck faeces		LPAI - H4N6,H5N1, H6N8		Nazir et al 2011
32-35	< 30 minutes	Chicken faeces		H5N1	sunlight	Songserm et al 2006
37	< 1 day	Chicken faeces		H5N1	dry and moist	Kurmi et al 2013

Temp (°C)	Duration of infectivity	Species / substrate	pH	AI virus strain / pathogenicity	Additional conditions	Reference
37	1-16 days	Chicken faeces		H7N2(considered LPAI)		Lu et al 2003
40	< 15 minutes	Chicken faeces	8.2	HPAI H5N1		Chumpolbanchorn et al 2006
42	<18 hours	Chicken faeces		HPAI H5N1	dry and moist	Kurmi et al 2013
56	15-30 minutes	Chicken faeces		H7N2(considered LPAI)		Lu et al 2003
-	<105 days	-	-	HPAI H5N2	wet and field conditions - no other details provided	Fichtner 1987
Litter / bedding						
10	4 days	Mixed wood shavings		LPAI	dry	Stephens et al 2017.
10	5 days	Mixed wood shavings		LPAI	moist	
16	4	Mixed wood shavings		LPAI	moist	
16	5 days	Mixed wood shavings		LPAI	dry	
21	2 days	Mixed wood shavings		LPAI	moist and dry	
27	1 day	Mixed wood shavings		LPAI		
10	3 days	Mixed wood shavings		HPAI	moist	
10	5 days	Mixed wood shavings		HPAI	dry	
21	2 days	Mixed wood shavings		HPAI	dry and moist	
32	1 day	Mixed wood shavings		HPAI	dry and moist	
43	1 day	Mixed wood shavings		HPAI	dry and moist	
21	5 days	Duck bedding		HPAI H5		
19-22	<60 hours	Boiler and turkey bedding	7.4-8.3	HPAI H5N8		Hauck et al 2017
19-22	<24 hours	Broiler, layer & turkey bedding	7.4-8.3	LPAI H6N2		Hauck et al 2017
25	1 day	Broiler litter - wood shavings	8-8.4	LPAI - H6N2,H5N2,H3N2		Reis et al 2012
25	1 day	Broiler litter - Wood shavings + peanut hulls	8-8.4	LPAI - H6N2,H5N2,H3N2		Reis et al 2012
25	<3 days	Broiler litter - Wood shavings and gypsum	8-8.4	LPAI - H6N2,H5N2,H3N2		Reis et al 2012
25	< 1 day	Chicken faeces	8.2	HPAI H5N1	UV light could not destroy the infectivity of the virus even after	Chumpolbanchorn et al 2006

Temp (°C)	Duration of infectivity	Species / substrate	pH	AI virus strain / pathogenicity	Additional conditions	Reference
					exposure for 4 hours.	
Compost						
35	6.4 hours ^b	Chicken manure and straw		HPAI H7N1		Elving et al 2012
45	1.7 hours ^b	Chicken manure and straw		HPAI H7N1		Elving et al 2012
55	29 minutes ^b	Chicken manure and straw		HPAI H7N1		Elving et al 2012
60	<15 days	Dead poultry and poultry waste		HPAI H5N1	Virus inactivated by composting when temp reached 60°C within 15 days	Ahmed et al 2012
Lake sediment						
0	66-394 days ^a	Lake sediment	7.4-8.4	LPAI-H4N6,H5N1,H6N8		Nazir et al 2011
10	43-54 days ^a	Lake sediment	7.4-8.4	LPAI-H4N6,H5N1,H6N8		Nazir et al 2011
20	13-18 days ^a	Lake sediment	7.4-8.4	LPAI-H4N6,H5N1,H6N8		Nazir et al 2011
30	5-11 days ^a	Lake sediment	7.4-8.4	LPAI-H4N6,H5N1,H6N8		Nazir et al 2011
Water						
17	94-158 days	Distilled water	7.4	LPAI H5/H7 & HPAI H5N1	Salinity 0 ppm	Brown et al 2007
17	83-182 days	Distilled water	7.4		Salinity 15 ppm	
17	82-115 days	Distilled water	7.4		Salinity 30 ppm	
28	26-30 days	Distilled water	7.4		Salinity 0 ppm	
28	28 days	Distilled water	7.4		Salinity 15 ppm	
28	17-18 days	Distilled water	7.4		Salinity 30 ppm	
-10	328-395 days	Lake surface water	7.84	LPAI H4N6, H5N1, H6N8	Salinity 150 ppm T-90 ^a : 66 days	Nazir et al 2010
-10	1399-1929 days	Normal saline	7.2		Salinity 9000 ppm	
-10	3454-3840 days	Distilled water	7.8			
0	188-208 days	Lake surface water	7.84		Salinity 150 ppm LPAI T-90: 35 days	
0	1013-1140 days	Normal saline	7.2			
0	2295-3347 days	Distilled water	7.8			
10	61-85 days	Lake surface water	7.84		Salinity 150 ppm LPAI T-90: 14 days	
10	353-419 days	Normal saline	7.2		Salinity 9000 ppm	
10	508-1185 days	Distilled water	7.8			

Temp (°C)	Duration of infectivity	Species / substrate	pH	AI virus strain / pathogenicity	Additional conditions	Reference
20	19-23 days	Lake surface water	7.84		Salinity150 ppm LPAI T-90: 4 days	
20	41-76	Normal saline	7.2		Salinity9000 ppm	
20	84-221 days	Distilled water	7.8			
30	13-14 days	Lake surface water	7.84		Salinity150 ppm LPAI T-90: 2 days	
30	14-35	Normal saline	7.2		Salinity9000 ppm	
30	30-77 days	Distilled water	7.8			
17	126-207 days (extrapolated from model)	Distilled water	7.3	H3N8, H4N6, H6N2, H12N5, H10N7	Duration varied with virus strain	Stallknecht et al 1990
28	30-102 days (extrapolated from model)	Distilled water	7.3	H3N8, H4N6, H6N2, H12N5, H10N7	Duration varied with virus strain	Stallknecht et al 1990
4	>30 days	Drinking water	6.7	HPAI H5N1		Yamamoto et al 2010
20	< 3 days	Drinking water	6.7	HPAI H5N1		Yamamoto et al 2010
21	>6 days		7.5	HPAI H5N8		Beerens et al 2021
-4	>32 days	River water		LPAI H7N2, H3N6		Webster et al 1978
22	>4 days	River water		LPAI H7N2, H3N6		Webster et al 1978
4	50 days	Freshwater lake surface water	6.95	H9N2		Zhang et a, 2014
16	17 days	Freshwater lake surface water	6.95	H9N2		Zhang et al 2014
28	5 days	Freshwater lake surface water	6.95	H9N2		Zhang et al 2014
Feathers						
4	240 days T-90:56 days	Chicken feather tissue		HPAI H5N1		Yamamoto et al 2017
4	160 days	Duck feathers		HPAI H5N1		Yamamoto et al 2010
10	73 days	Duck feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
10	38 days	Emu feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
10	42 days	Duckling feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
20	15 days	Duck feathers		HPAI H5N1		Yamamoto et al 2010
20	30 days	Chicken feather tissue		HPAI H5N1		Yamamoto et al 2017

Temp (°C)	Duration of infectivity	Species / substrate	pH	AI virus strain / pathogenicity	Additional conditions	Reference
	T-90:10 days					
25	45 days	Duck feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
25	17 days	Emu feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
25	18 days	Duckling feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
37	30 days	Duck feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
37	7 days	Emu feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
37	9 days	Duckling feathers		HPAI H5N1	High viral load	Karunakaran et al 2019
Metal						
0-4	<2 days	Galvanised steel metal		HPAI H5N1	Low humidity 25-30% & UVA/B	Wood et al2010
4-7	13 days	Galvanised steel metal		HPAI H5N1	Low humidity 15-46%	Wood et al2010
7-8	<9 days	Galvanised steel metal		HPAI H5N1	High humidity 79-97%	Wood et al2010
22-23	<1 days	Galvanised steel metal		HPAI H5N1	High & low humidity	Wood et al2010
Room T	>3 days	steel				Tiwari et al 2006
Various materials						
0-4	<1 day	Glass		HPAI H5N1	Low humidity 25-30% & UVA/B	Wood et al2010
0-4	< 4 days	Soil		HPAI H5N1	Low humidity 25-30% & UVA/B	Wood et a.2010
4-7	13 days	Glass Soil		HPAI H5N1	Low humidity 15-46%	Wood et al2010
4-7	< 13 days	Glass Soil		HPAI H5N1	High humidity 79-97%	Wood et al2010
23	<1 day	Glass Soil		HPAI H5N1	High & low humidity	Wood et al2010
25	26 hours	Plastic		HPAI- H5N1	Humidity 45-55%	Bandou et al 2022
25	10 hours	Plastic		HPAI- H5N3	Humidity 45-55%	Bandou et al 2022
25	<10 hours	Plastic		HPAI – non H5N1/H5N3	Humidity 45-55%	Bandou et al 2022
25	4.5 hours	Human skin		HPAI -H5N1	Humidity 45-55%	Bandou et al 2022
25	~ 2hours	Human skin		HPAI- non H5N1	Humidity 45-55%	Bandou et al 2022
19-22	>72 hours	Footbath		HPAI H5N8, LPAI H6N2	Quaternary ammonia	Hauck et al 2017
19-22	>72 hours	Footbath		HPAI H5N8, LPAI H6N2	Quaternary ammonia =	Hauck et al 2017

Temp (°C)	Duration of infectivity	Species / substrate	pH	AI virus strain / pathogenicity	Additional conditions	Reference
					glutaraldehyde	
19-22	0 hours	Footbath		H5N1, H5N8, LPAI H6N2	Chlorine granules (bleach)	Hauck et al 2017

a T-90 values represent the time required for 90% loss of virus infectivity as calculated by linear regression modelling
b time to reach 12-log₁₀ reduction of virus

Appendix 4 Control measures following detection of AI in wild birds only

Table A4.1 Summary of control measures to be applied to poultry or captive birds and wild birds following detections of HPAI and LPAI in wild birds only

	Disease subtype			
	HPAI in wild birds only – controls on wild birds	HPAI in wild birds only – controls on poultry and captive birds	LPAI (H5/H7) in wild birds only – controls on wild birds	LPAI (H5/H7) in wild birds only – controls on poultry and captive birds
Control measure				
Stamping out of infected birds	No	N/A	No	N/A
Enhanced biosecurity on poultry and captive birds ^a	N/A	Yes	N/A	+/- ^c
Declared areas	+/-	+/-	No	No
Movement controls	No, not possible ^d	+/-	No	No
Tracing and surveillance	Surveillance only; tracing not possible	Surveillance only; tracing not applicable	Baseline surveillance continues; tracing not possible	Baseline surveillance continues; tracing not applicable
Vaccination ^b	+/-	+/-	No	No
Pre-emptive destruction	No	No	No	No
Disposal of dead birds and waste	+/-	N/A	No	N/A
Decontamination of environment	No	No	No	No
Public awareness campaign	+/-	+/-	+/-	+/-

+/- = control measure may or may not be used, subject to risk assessment; HPAI = high pathogenicity avian influenza; LPAI = low pathogenicity avian influenza

a Biosecurity controls should be in place on an ongoing basis because wild birds are reservoir hosts for AI viruses in Australia

b Depends on limiting factors, including vaccine approval and availability

c Subject to risk assessment

d Movement controls may be applied to other items (e.g. waste, vehicles, equipment, carcasses etc.)

Glossary

Terms and definitions

Standard AUSVETPLAN terms

For definitions of standard AUSVETPLAN terms, see the **AUSVETPLAN Glossary**.

Manual-specific terms

Term	Definition
Captive birds	Any bird (other than poultry species) that is kept in captivity, including but not limited to those that are kept for shows, exhibitions, zoological collections, or competitions, as well as pet birds, and those kept for breeding and/or selling. This includes wild birds that are temporarily in captivity under the dedicated care of a wildlife carer.
Clade	A further classification of influenza viruses based on the similarity of their HA gene sequences (WHO/OIE/FAO H5N1 Evolution Working Group 2008)
Controlled marketing	Orderly marketing of birds through their normal production cycle that have been shown to be not infected and to which any risk for disease spread has been mitigated by appropriate biosecurity measures.
Cyanosis (adj: cyanotic)	Blueness of the skin and/or mucous membranes due to insufficient oxygenation of the blood.
Egg marketing premises	Premises where table eggs are graded and packed for the retail market. The premises may also contain a pulp plant and facilities for manufacture of egg-based products.
Egg pulp	A homogeneous liquid made from either whole liquid egg, egg albumen or egg yolk, pasteurised for marketing as a liquid or frozen product.
Empty period	The time between decontamination and restocking of premises with sentinels or commercial restocking.
Further processing plant	A plant that receives fresh carcasses from an abattoir for cutting up; processing into poultry nuggets, rolls and so on; and cooking or partial cooking for fast-food outlets and retail markets.
Galliformes (adj: gallinaceous)	The order of birds that includes poultry, turkey, pheasant and peafowl.
Haemagglutination	Agglutination of red blood cells by a specific antibody or other substance.
Haemagglutinin (vb: haemagglutinate)	Protein on the virus surface that agglutinates red blood cells.
Modified stamping out	Process in which stamping out is undertaken, but delayed or staged until logistically feasible. It may be used in combination with suppressive emergency vaccination strategies where the goal is to suppress virus replication in high-risk animals by using emergency vaccination and then depopulating vaccinates.

Term	Definition
Pathogenicity	Competence of an infectious agent to produce disease in the host species. The relative disease changes are described as highly, mildly or lowly pathogenic. Nonpathogenic describes the situation where infection produces no disease or clinical signs in a susceptible host. <i>See also</i> Virulence.
Peracute	Extremely acute form of a disease.
Poultry	For the purposes of this manual, 'poultry' means chickens, turkeys, guineafowl, ducks, geese, quail, pigeons, pheasants, partridges, emus and ostriches reared or kept in captivity.
Poultry (commercial)	All birds reared or kept in captivity for the production of any commercial animal products (or for breeding for this purpose), and all birds used for restocking supplies of game (or for breeding for this purpose), until they are released from captivity.
Poultry (non-commercial)	Chickens, turkeys, guineafowl, ducks, geese, quail, pigeons, pheasants, partridges, emus and ostriches that are kept in captivity for purposes other than production of commercial animal products, restocking supplies of game, and/or breeding for these purposes.
Pre-emptive destruction	Destruction of animals at high risk of infection but in which infection has not yet been demonstrated. Birds do not go for human consumption.
Process slaughter	Slaughter of animals for human consumption after they have been transported, under movement controls, to a processing plant.
Processing plant	An abattoir for slaughtering animals for human consumption, with chilled and frozen storage facilities.
Proventriculus	The front (thin-walled) part of the stomach in birds.
Psittaciformes (adj: psittacine)	Parrots and related groups of birds.
Rendering	Processing by heat to inactivate infective agents. Rendered material may be used in various products according to particular disease circumstances.
Virulence	Capacity of an infectious agent to produce pathological changes. Agents that do not produce any disease signs are described as nonvirulent or avirulent. <i>See also</i> Pathogenicity
Wild birds	Any avian species that are free living in their natural environment without direct human intervention or control. This includes native and exotic/introduced bird species.
Zoo birds	Captive birds that are maintained at a zoo premises.

Abbreviations

Standard AUSVETPLAN abbreviations

For standard AUSVETPLAN abbreviations, see the **AUSVETPLAN Glossary**.

Manual-specific abbreviations

Abbreviation	Full title
AI	avian influenza
EID	egg infectious dose
HA	haemagglutinin
HPAI	high pathogenicity avian influenza
LPAI	low pathogenicity avian influenza
NA	neuraminidase
PPE	personal protective equipment
TCID	tissue culture infective dose

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